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*Levine, S Z.: J.A.M.A. 128:283, May 26, 1945

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*Kamman, G. R.: Fatigue as a Symptom in Depressed Patients, Journal-Lancet 65:238 (July) 1945.

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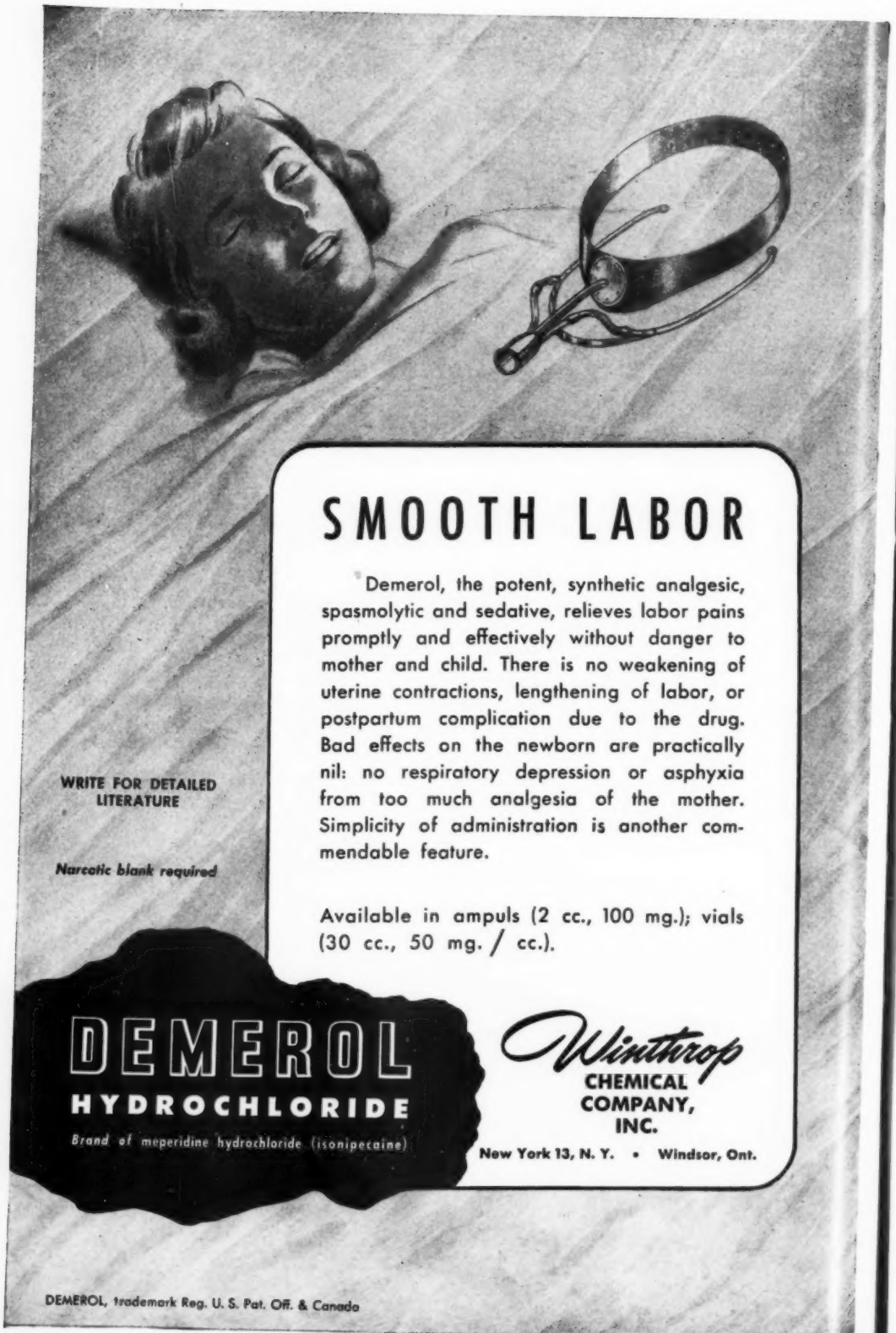
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(1) Manolew, E. E., Jr., and Atwell, R. T.: South. M. J. 39:726 (Sept.) 1946.
(2) Woodward, F. D., and Hein, T. J.: J.A.M.A. 129-589 (Oct. 27) 1945.

PENICILLIN SCHENLEY. Suggested dosage: Intramuscular, 20,000 to 40,000 units every three hours, continued until the patient has been symptom-free for forty-eight to seventy-two hours. Topical, instillation of 3 to 5 cc. of a solution containing 5,000 to 10,000 units per cc., repeated as frequently as indicated in the judgment of the physician.

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SCHENLEY LABORATORIES SERVICES: 1. Penicillin Paragraphs for March, dealing with upper respiratory infections, has been mailed to all physicians. 2. A comprehensive penicillin dosage chart will be mailed to physicians on request.

NO. 9 in Schenley Laboratories' continuing summary of penicillin therapy

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Striking and hitherto unsuspected protein loss has been observed in patients with fractures. Excessive urinary nitrogen excretion reaches its maximal point about a week after the injury is sustained, and thereafter slowly diminishes in extent, so that nitrogen balance is restored in approximately four weeks.[1]

In patients sustaining severe burns, the daily protein loss may be equivalent to 400 cc. of plasma.[2]

In a study embracing 23 burned patients, nitrogen balance determinations revealed excessive urinary nitrogen excretion. Nearly all patients were in negative nitrogen balance which was most marked during the first ten days.[3]

It thus appears that protein destruction and loss are prominent and potentially detrimental sequelae of trauma, and that every effort must be made to restore nitrogen equilibrium as quickly as possible to prevent the many deleterious consequences of protein depletion. The recommendation has been voiced that "whenever possible, protein losses or deficiencies should be corrected by oral feeding."[4]

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---

1 Howard, J. E.: Bull. Johns Hopkins Hosp., *74*:313 (May) 1944.

2 Co Tui, C.; Wright, A. M.; Mulholland, J. H.; Barcham, T., and Breed, E. S.. Ann. Surg. *119*:813-823 (June) 1944.

3 Hirshfeld, J. W.; Abbott, W. E.; Pilling, M. A.; Heller, C. G.; Meyer, F.; Williams, H. H.; Richards, A. J., and Obi, R.: Arch. Surg. *50*:194 (Apr.) 1945.

4 Lund, Chas. C., and Levenson, S. M.: J. A. M. A. *128*:95 (May 12) 1945.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

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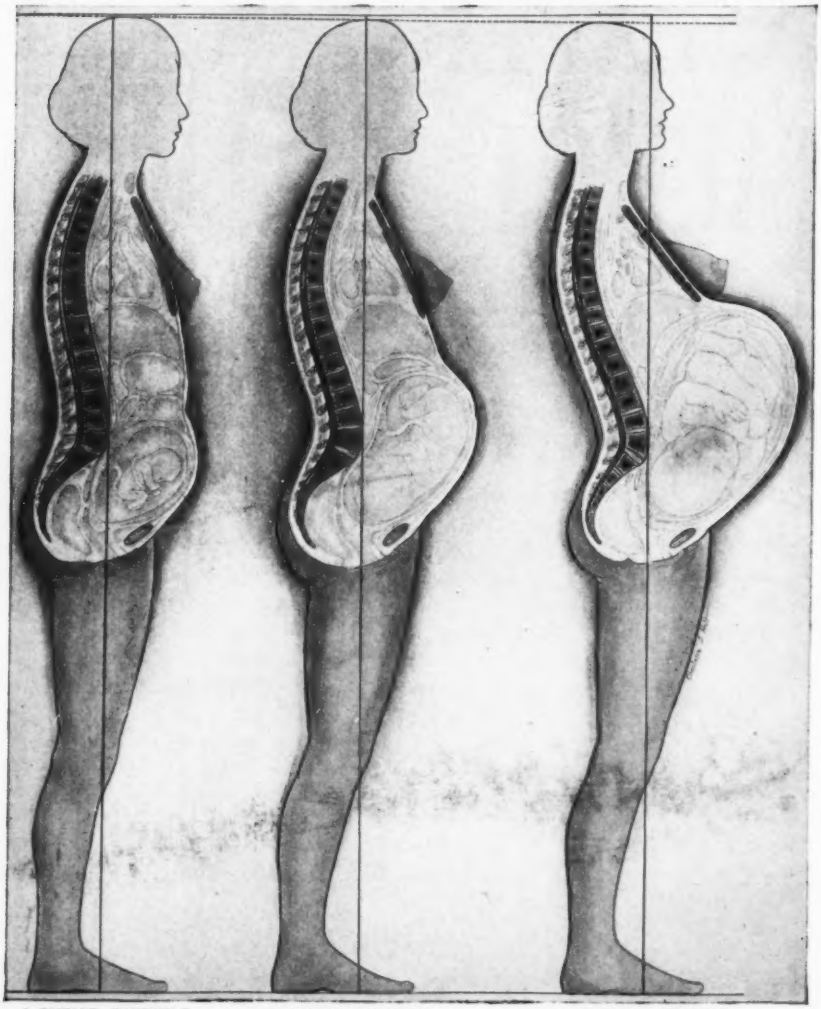
BODY MECHANICS OF PREGNANCY

• STUDIES FROM LIFE MODELS DURING PREGNANCY • Illustration by Charlotte S. Holt



4 LUNAR MONTHS 7 LUNAR MONTHS 10 LUNAR MONTHS

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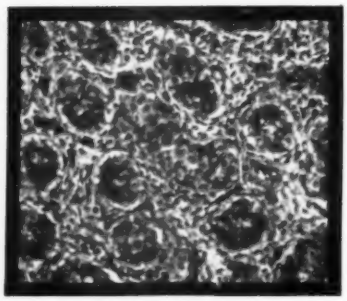

Photomicrograph of undescended human testicle. Transection of tubules. Masson III stain.

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1. Thompson, W. O.: J.A.M.A., 132: 185, 1946.

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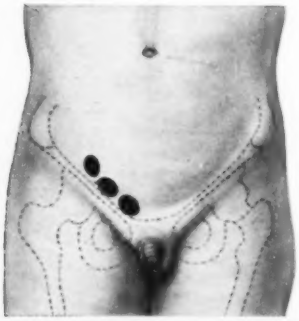

MALPOSITION OF THE TESTICLE

Right abdominal testicle. Three positions of the undescended testicle in the abdomen.

Right pubo-scrotal testicle. Position of the undescended testicle in the pubo-scrotal region.

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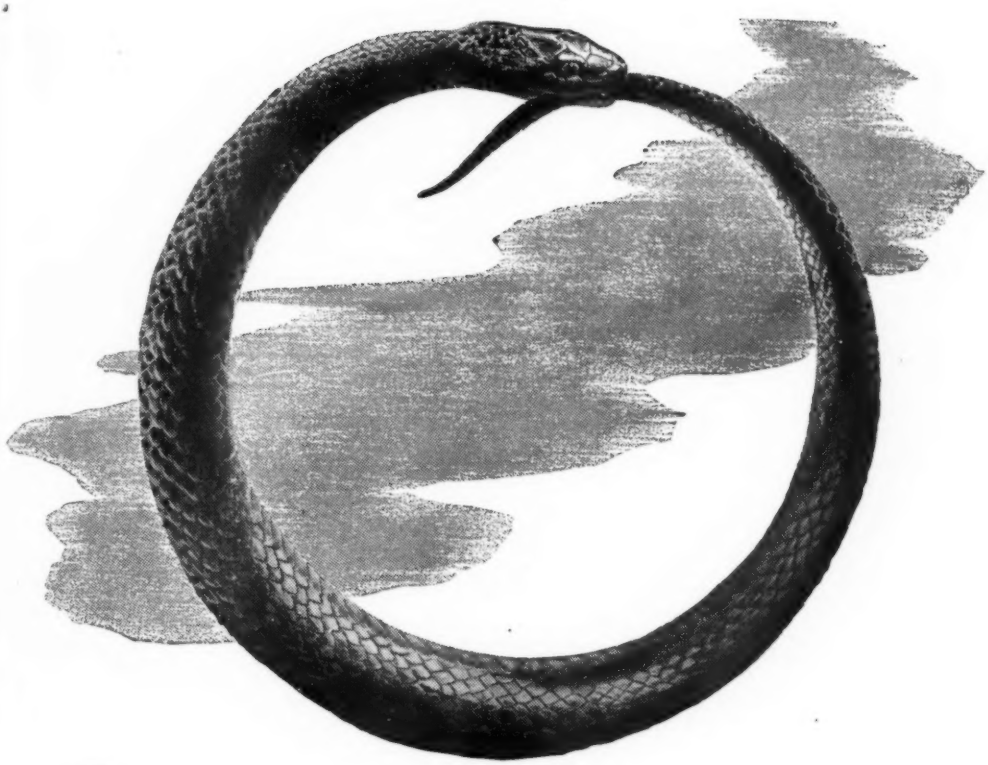

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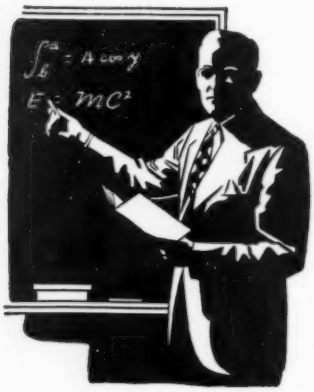

Molder of Minds . . . . .

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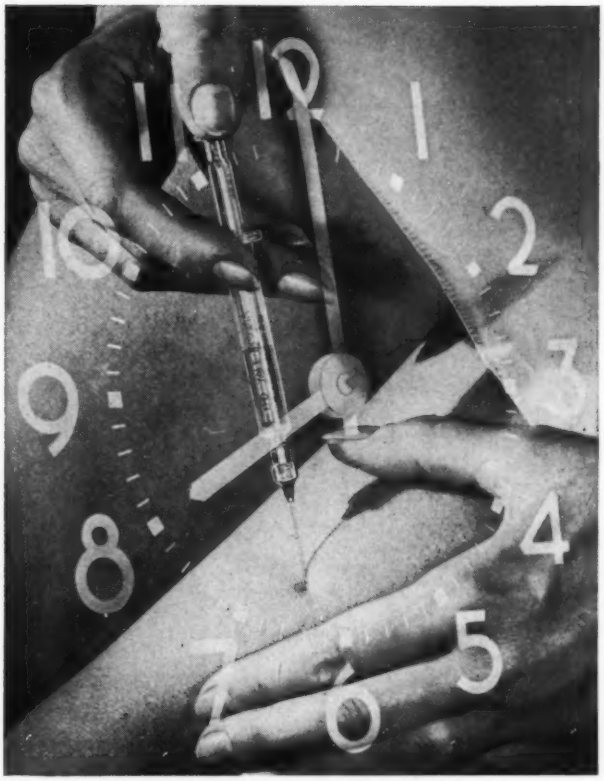
*Based on average reported values for milk.

One-injection control of diabetes



THE LIFE OF MANY DIABETICS, complicated by the need for two, and sometimes three, daily injections of insulin, can be simplified by a change to 'Wellcome' Globin Insulin with Zinc—which, because of its intermediate action, may provide adequate control with *only one injection a day.* This welcomed change-over can be made in three clear-cut steps:

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2. ADJUSTMENT TO 24 HOUR CONTROL: Gradually adjust the Globin Insulin dosage to provide 24-hour control as evidenced by a fasting blood sugar level of less than 150 mgm. or sugar-free urine in the fasting sample.

3. ADJUSTMENT OF DIET: Simultaneously adjust carbohydrate distribution of diet to balance insulin activity; initially 2/10, 4/10 and 4/10. Any midafternoon hypoglycemia may usually be offset by giving 10 to 20 grams of carbohydrate between 3 and 4 p.m. Base final carbohydrate adjustment on fractional urinalyses.

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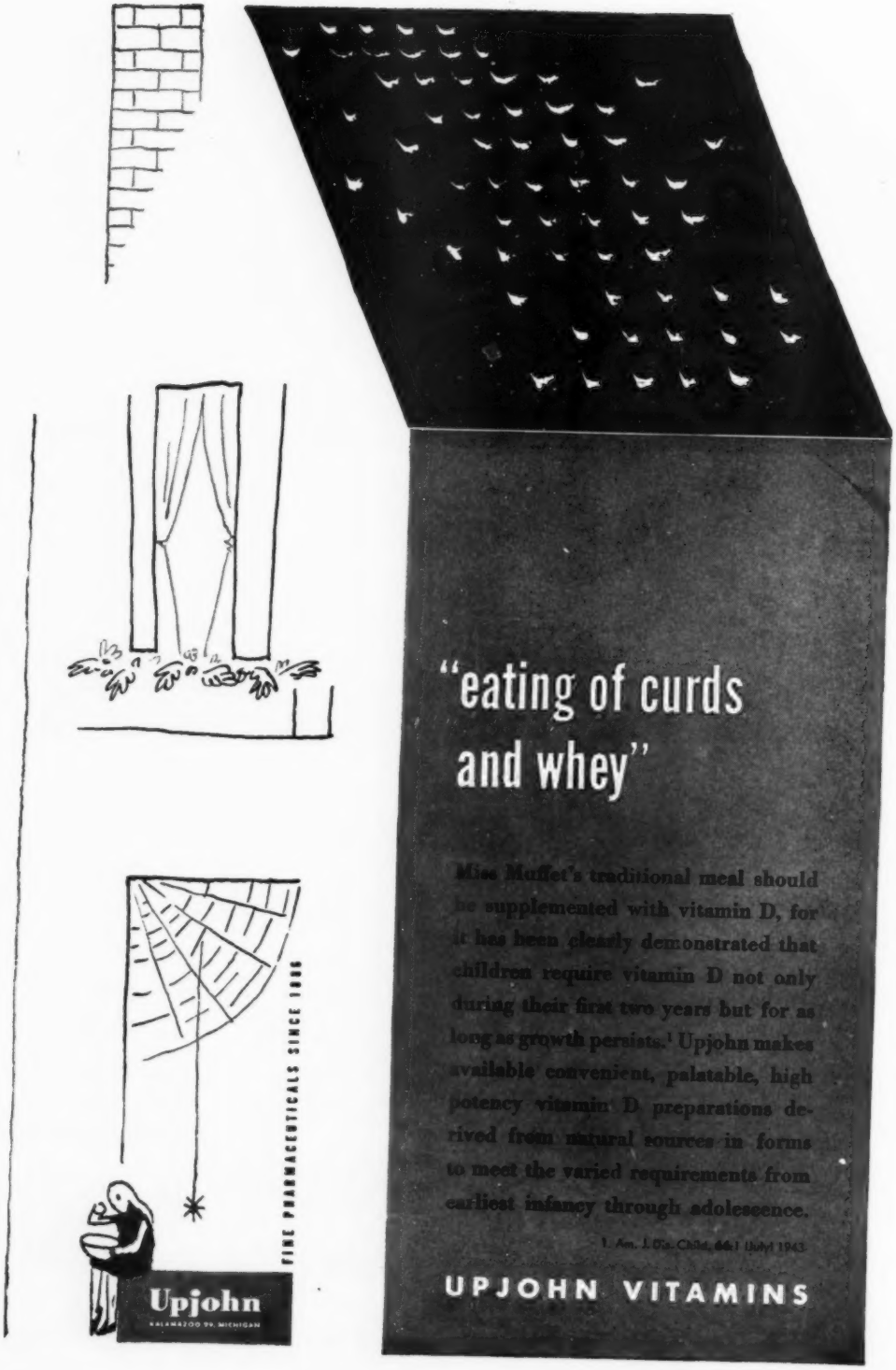
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1. Am. J. Dis. Child. 66:1 (July) 1943
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THE consensus of clinicians who have had considerable experience with aurotherapy is that gold, despite its recognized toxicity, is the most effective agent available for the treatment of active rheumatoid arthritis.

The following statements, quoted from the article entitled, "The Use And Abuse Of Gold Therapy In Rheumatoid Arthritis," by Bernard I. Comroe, M. D. (*J.A.M.A.* **128**:848-851, July 21, 1945), constitute an excellent summary of the present position of gold therapy in arthritis:

1 Gold is of no value in any form of joint disease except rheumatoid arthritis.

2 Gold does not benefit all patients with rheumatoid arthritis.

3 Gold is not the final answer to the treatment of rheumatoid arthritis.

4 Toxic symptoms may appear at any time during this form of therapy.

5 From 10 to 20 per cent or more of patients who have received gold therapy relapse after stopping the drug.

6 Extreme care must be used during gold therapy, and the physician must be familiar with the details of such treatment before undertaking this.

7 Injections of certain gold salts in proper dosage may be followed by subjective and objective evidence of improvement in the majority of selected patients with rheumatoid arthritis.

Literature on request

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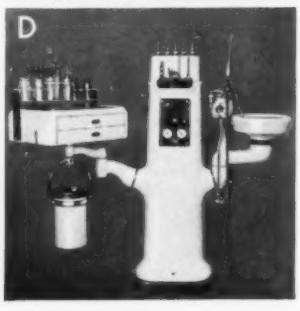

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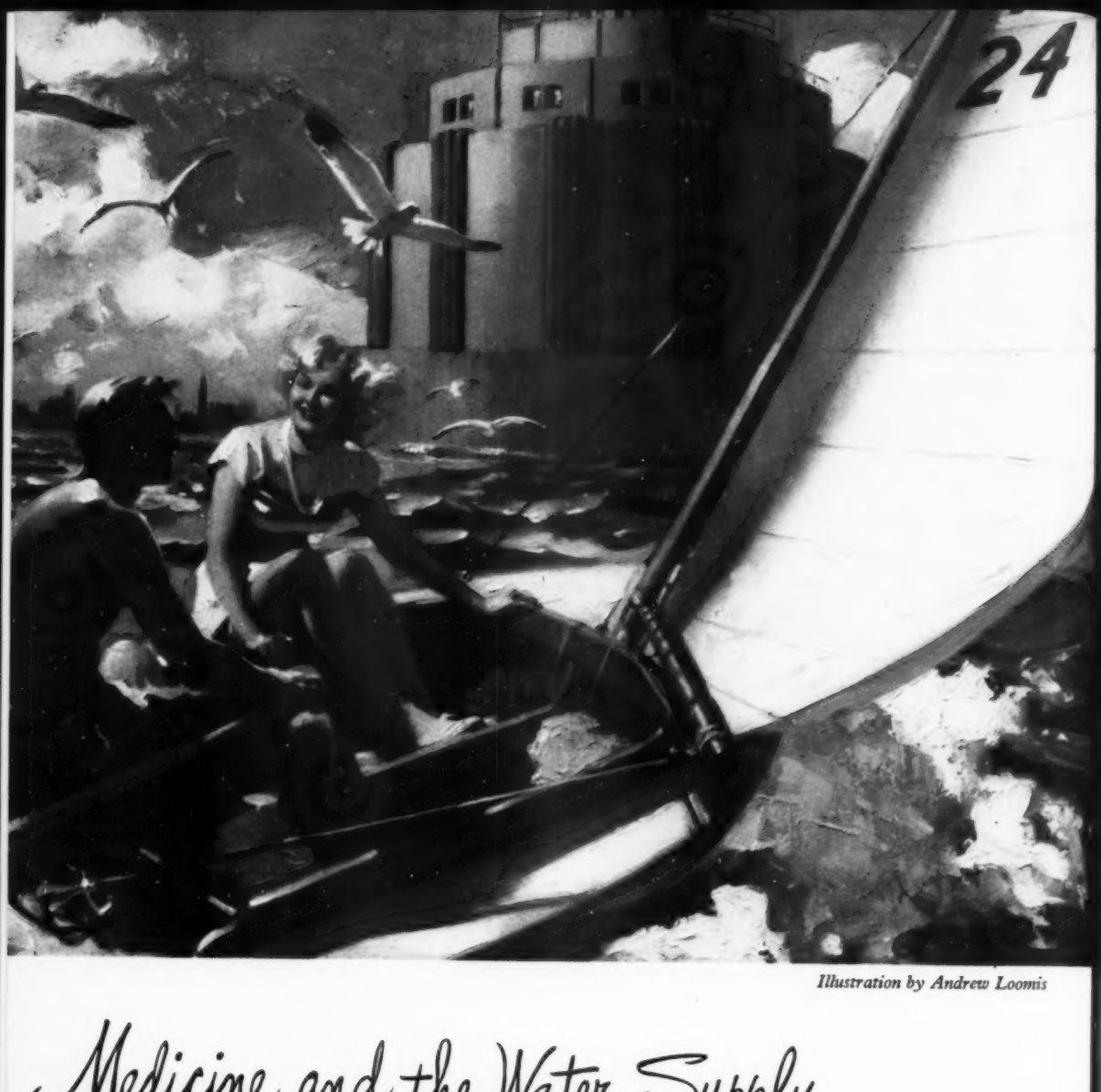

Illustration by Andrew Loomis

Medicine and the Water Supply

Preventive techniques have been the concern of the ethical pharmaceutical manufacturer as well as of the physician. The Lilly Research Laboratories devote a generous amount of time and effort to fields of pure research. In addition, scholarship and research grants are made regularly to recognized medical schools.

BEHIND THIS Lake Michigan pumping station, just offshore at Chicago, lies the city's famous sky line. Here, within the metropolitan area, almost four million people live in close proximity to one another. So pure is the water supply that from the millions of city water outlets one may drink with no fear of contracting a water-borne disease. Water purification is one of the many advancements, frequently taken for granted, for which medical science is chiefly responsible. Through methods worked out by the physician and the engineer, people may dwell together in large cities in complete freedom from fear of water contamination.

NEWER ASPECTS OF PANCREATIC DISEASE

FELIX CUNHA, M. D.
San Francisco, California

I AM one of the novices here. I cannot help but take out a moment to express my sincere gratitude to Doctor Salsbury for having invited me to come here. I have the advantage of having been here now for three days during which the tranquility, the freedom from tension, the quiet efficiency with which things are done have made a deep impression on me. Being to no mean extent ranch and ranch products minded, the self sufficiency of this oasis in the desert is truly amazing. Particularly are these things truly more impressionistic to one who for years has summed up his philosophy of living in the following words.

This journey through life is similiar to a long one-way railroad trip, you are privileged to make as many stop-overs as you choose, you may take several detours, but there is no return trip. This visit to Ganado represents one such stop-over and one which will not soon be forgotten.

This morning I am in a sort of numerical or arithmetical daze. Dr. Phil Thorek has told you how in diagnosing an acute abdomen, there are eighty-six different pathological possibilities within the adbomen that have to be considered in a differential diagnosis.

Following this Doctor Rigg told you that there are two hundred and seventy-nine causes of headaches. Dr. Max Thorek cites 4664 or was it 4464 cases in which he has done obliteration of the gall bladder by electro-coagulation. Sanwiched in here somewhere were some remarks about the simplicity of gastric resection, as reviewed by some author in a recent journal, with no deaths in one hundred consecutive cases or was it two deaths in two hundred twenty cases. Please recall that I prefaced these statistical citations by saying that I was in an arithmetical daze and I still am.

Before going into the subject of pancreatic disease I would like to take the liberty of asking a question of Dr. Orr with regard to the incidence of a higher mortality in males following choleeystectomy. I believe one or two other speakers mentioned the same experience. Could this be explained on the basis of an interference in liver function, particularly in the inability of the liver to store glycogen brought about by a moderate alcohol intake over a long period of time, not a cirrhosis, not even a pre-cirrhotic state, but lacking any truly descriptive term, a sub-clinical hepatitis, yet not thinking of hepatitis in any way as replacement or loss of tissue, but in the main as a disturbance of liver glycogen reserve.

The second divergence from the subject I would like to take the liberty of making, would be to ask Dr. Phil Thorek a question. In his remarks on perforated peptic ulcer, he gave as his own experience and that of others, that it is an exceedingly rare occurrence in females. I would agree with him whole-heartedly—up to eighteen months ago. Since then I have seen three cases, the last one only two months or so ago. What at that time was most striking about each and all of them, was that at no time, did they simulate the classical picture we have been taught to look for in perforated ulcer.

True, they had pain, epigastric pain, sharp, severe, but they were not prostrated, they were not in shock—each was able to leave her work in a downtown office building, get into a taxi and get to the hospital under her own power. The pulse rate was only moderately elevated, temperature was normal or slightly under normal, blood pressure levels unaffected.

Phases of ulcer conformity and activity seem to follow a hills and valleys pattern somewhat similar to a fever chart in an intermittant fever.

Reminiscing for but a moment to cite a possible analogy to this recent more frequent occurrence of ulcer perforation in the female—about eighteen years ago, Prefessor Finsterer had in Vienna as his honored guest for a few days, Dr. Will Mayo. It so happened that during the days of Dr. Mayo's visit, the predominence of the ulcers resected were unusually large, penetrating deeply into the gastrohepatic ligament, some into the liver substance itself, and most of such size, that without exaggeration, you could put your whole fist into their craters without difficulty, and without stretching either the imagination or the ulcer. Dr. Mayo was evidently disturbed and on the day of his last visit to the clinic drew the Americans present aside and

FOOT NOTE: Presented before the Harlow Brooks Memorial Navajo Clinical Conference. Sage Memorial Hospital, Ganado, Arizona, Aug. 29, 1946.

said, "Don't go home with the idea that the ulcers you have been seeing here are what you will meet in America. Not so. Our ulcer is small, the size of a dime usually, cleanly punched out etc., etc. You don't have to be so radical in your handling of them, as they are here."

In the course of time, behold, the men in America began to see more and more of the large, deep, penetrating ulcer and the men in Europe more of the small, sharply outlined, clear cut shallow ulcer. No wonder confusion existed—the European surgeon and the American surgeon were discussing peptic ulcer, but each with a different mental conception of what he was talking about. To go back to the point of divergence, could it be that having just passed thru a period where the female has been subject to unusual stress and strain, more than ever before we may be on the threshold of a period wherein we will see more ulcer perforatons in the female? Time alone will answer.

I noted that not one or two of the previous speakers but several played around with more or less facetious and disparaging remarks about the age of forty. That is definitely very dangerous ground. May I call to your attention that the great Osler got into extremely hot water on that subject and was truly never able to live it down. Just a word of caution.

The work on pancreatic disease cited here was started before the war as a research problem. Very soon it established three distinct phases or avenues along which studies should proceed.

The first phase was that of acute inflammatory disease of the pancreas. In this phase we believe we did obtain some degree of success in establishing certain helpful criteria for the handling of these cases and the establishment of certain laboratory procedures as being distinctly helpful in keeping the surgeon informed as to the status, progress and prognosis of his case. It is these I wish to present here.

The second phase of these pancreatic studies was concerned with the secretory activity of the gland, an endeavor to establish normal limits of trypsin, amylopsin and lipase values under normal conditions, and then under conditions in which a sub-acute or chronic pancreatic dysfunction is suspected. More specifically, we were concerned with trying to establish a "pancreatic achylia" as a definite clinical entity, somewhat similiar to a gastric achylia, using secretin to stimulate pancreatic secretion in the same manner we use histamine to provoke gastric secretion.

Into these studies we tried to introduce the concept of "pancreatic asthenia". Not the severe down hill asthenia as described by Whipple as a sequel to acute pancreatitis, but as an intermittent sub-clinical entity and we tried to tie it in with either an achylia of pancreatic secretion or with disturbance of sugar metabolism or insulin stability.

We tried to establish that such a pancreatic achylia need not necessarily be constant or permanent but could be intermittent, and "intermittent pancreatic achylia."

Unfortunately with war time these studies had necessarily to be postpones and our attention focussed on the more material and concrete problems of civilian needs. However we are hopeful that these studies will shortly be resumed.

As to the third phase of these studies it concerned itself mainly with trying to establish the concept of chronic pancreatic disease as a definite clinical diagnosis, to furnish a lead to the puzzled medical man who finds himself faced with a patient complaining of gastro-intestinal distress not conforming in any way to any pattern he knows and wherein exhaustive studies have all been negative including roentgenography of the gall bladder.

It was also slanted toward explaning—partially at least Osler's six and one half line description of the pathology of chronic pancreatic disease back in 1890.

"The organ is firmer than normal, the interstitial connective tissue is increased, *and there is more or less change in the excreting structures. There may be marked pigmentary changes. Degeneration of the glandular elements is present in these cases.*" Osler's great reputation was founded upon a clear concise description of what he saw.

Chiari in Vienna, also Erdheim, described a sclerosis of the pancreas in about the same words with the addition that they emphasized the size of the gland, its hardness, its contraction, its smallness.

This third phase of study was also necessarily postponed because of sudden inaccessibility to material due to war time conditions.

To return to the first phase and report as to what actually was learned of material value to others as well, we turn to a short description of the acute phase of the disease.

How is an acute pancreatitis produced. There are many ingenious theories and explanations.

There has always been an aura of mystery surrounding the pancreas. Many of the great names in medicine are associated with studies of its functions. Claude Bernard, de Graaf, Langerhans, Fitz of appendicitis fame, Pavlov, and more recently Banting and Best are only a few.

Osler described the symptomatology, and under pathology, the fat necrosis of the acute disease. He commented upon the possibility of suppurative pancreatitis and reported thirty-seven cases all told of pancreatic disease. To the subject of chronic pancreatitis he devoted a total of six and one-half lines, based apparently on postmortem observation alone and stated his ideas as in the quote above.

Amplifying this slightly a few years later he said there may be marked pigmentary changes; a similar condition has been found in the liver. Degeneration of the glandular elements is present in these cases. The sclerosis may be associated with calculi in the ducts

Today the accurate diagnosis of sub-acute and chronic pancreatic disease still presents one of the difficult problems of medicine. Because of its extreme variance in symptomatology and because of the inadequacy of our ways and means of obtaining more specific knowledge of pancreatic secretion and function.

In spite of this inadequacy, clinical experience has taught us how to manage these cases better. In spite of the fact that cases of acute pancreatic disease are not common, yet we are able to say, not as Osler said fifty years ago, that these cases are uniformly fatal, but that a respectable percentage of patients are able to recover from the disease.

Mechanical factors described as playing a part in the production of acute pancreatic disease are all based upon the concept that some occlusion takes place in the pancreatic ducts, either the main duct alone or along with the accessary duct, thereby preventing the enzymatic juices from reaching the duodenum and converting the open duct system into a closed one, the enzymes being forced to back up into the pancreas itself and a process of autolysis initiated. Without such an occlusion, reflux of bile along the pancreatic ducts into the pancreas substance has also been described as an etiological factor.

Other physical factors acting to bring about changes in the pancreas might be a gallstone in the duct of Wirsung or in the accessory pancreatic duct, a gallstone at the ampulla of Vater, spasm of the sphincter of Oddi, or edema of the duodenum in the region of the ampulla of sufficent degree to cause occlusion of the common bile duct and turn back the normal onward flow of bile at this point, so that the bile backing up the common duct, meeting resistance from behind is forced into the pancreatic ducts or duct, then into the substance of the pancreas where by its irritant action, it produces those changes we associate with pancreatitis. However, some evidence has accumulated that it is possible for a common channel mechanism to exist carrying simultaneously both bile and pancreatic enzyme without harm to pancreatic tissue.

Up to the present time, bile has borne the brunt of blame as being the most important single element in the causation of acute pancreatic necrosis. The theories have all been that bile getting outside of its normal physiological habitat and getting into a place wherein it is a foreign invader or into a field physiologically unadapted to resist the invader or accomodate itself to its presence, has caused tissue destruction in the pancreas. Much experimental data is favorable to the acceptance of this theory. It is well known that in the experimental animal, injection of bile into the pancreatic ducts causes extensive necrote destruction of pancreatic tissue. This experiment has been repeated on endless number of times but does not seem to lead anywhere in helping to interpret pancreatic destruction in the living human being.

A multitude of experiments demonstrating the action of trypsin on different types of tissue have been performed to demonstrate a possible involvement in etiology. By some, trypsin has been considered an actual malefactor. The liberation of this ferment of highly concentrated digestant action outside of its normal confines is present in all cases of acute pancreatic necrosis, but what factor changed the controls that it is enabled to escape outside its normal habitat is not known.

Most poignant of all this speculation is the contention that normal bile backing up from the bile system into the pancreas will not produce pancreatic necrosis, that only infected bile, such as would be found in an acute, sub-acute, or chronic inflammatory process in the gall bladder or duodenum will produce pancreatic change

and that such change occurs by way of abnormal secondary trypsin activation.

This possible symbiotic action of bile and trypsin, or infected bile and trypsin together should not be overlooked. It has been demonstrated that the toxic effect of bile can be increased by the presence of proteolytic agent such as trypsin could be, acting as an advance guard, digesting any mucus or serum proteids which might be in the way or acting as a defending barricade. Such types of substance are known to inhibit the action of bile. Trypsin digestion of such proteids would leave the field clear for a more concentrated and devastating action of bile.

Evidence has been produced to explain the frequent association of gall bladder disease with pancreatitis on the basis of direct extension of infection from the gall bladder to the pancreas by way of a rich system of lymphatics. This same process would hold true as in the manner of extension of infection from an inflammatory gastric or duodenal lesion, most freguently ulcer, as the anatomical proximity to the pancreas is common to all. The fact that in early acute pancreatitis the head is the part most frequently involved, and as the head just directly into that retroperitoneal area most rich in lymphatics, this observation is offered corroborative of this evidence. The part played by duodenal diverticulae situated at the ampulla of Vater has not been studied sufficiently. Here it would be possible for bile to stagnate, produce an irritative edema, become infected and produce a secondary inflammatory edema, obstruction of the common duct and reversal of the biliary flow. Considering the increasingly large number of duodenal diverticulae which are being demonstrated at this site, through or because of improved methods of roentgenography, speculating upon such a possibility, although found admittedly infrequent, is justified. Unfortunately, the surgeon operating upon a patient with acute pancreatitis, in fairness to the patient's chances of recovery, has not the time for extensive exploration to determine whether or not a diverticulum at the ampulla is present. Such exploration would hardly be justified and the difficulties attending the actual identification and exploratory examination of such a diverticulum even if one were present should not be minimized.

Embolism of the pancreatic artery or one of its branches can also be taken into consideration as a possible etiological factor in the production of acute pancreatic disease. Along the same line of reasoning as that in production of ulcer, a small sections of pancreatic substance from their blood supply with ensuing production of l o c a l necrosis, extension of this necrosis, et cetera, might well be the mode followed.

In passing one might mention the association of acute hemorrhagic changes in the pancreas with acute alcolholism as verified fairly commonly at autopsy. That some pancreatic changes, particularly as shown by changed blood sugar levels takes place, is evidenced clinically by the cases which following an alocholic bout complain of symptoms consistent with hypoglycemia.

III—SYMPTOMS OF ACUTE PANCREATITIS

For the past forty to fifty years the standard textbooks of medicine have been describing in a rather melodramatic way the symptoms of acute hemorrhagic pancreatitis. Invariably, the symptoms of shock and collapse have been overemphasized, similarly to the old descriptions of perforated peptic ulcer, in neither of whch type of case is this essentially true. Such descriptons occur so systematically repeated that one wonders if the author is of such supposedly authoritative sources of information do not lift some previous work and change here and there some wording or grammar, inject a personal observation, but either due to lack of time or sincerity, do not in truth produce a work wherein a disease description is brought completely up to date in the light of contemporary individual experience.

Pain is of course the paramount symptom. It is sudden in onset, steady, continuous, without remission, located focally just above the umbilicus, usually transmitted straight through to the back, to serve to confuse us more in our differential diagnosis of perforated duodenal ulcer, rupture of the gall bladder, stone in the common duct and what not. Clinicians have studied the radiation of this pain. Statisties are offered and the consensus is that radiation straight through to the back is one of the almost constant features of the pain of acute pancreatic disease.

When one has menioned pain, sudden in onset, localized in an area just above the umbilicus, severe, sharp, radiating through to the back, some degree of nausea and vomiting and then some degree of prostration and shock, one has covered about all that is present in acute pancreatitis, but, unfortunately, the same symptom

complex describes a great many other acute upper abdominal conditions.

Earlier writers stressed the fact that the pain was so severe that it did not lend itself to control by morphine but such a statement deserves some degree of qualification and unfortunately such a phenomenon is present also in other upper abdominal emergencies, so that the point is of little value in differential diagnosis.

On physical examination nothing specifically objective presents itself to help in differential diagnosis. Perhaps the most important point that can be stressed is that these patients are not in a state of extreme shock or collapse as the older writers and textbooks were so apt to paint the picture.

The mere fact that in a high percentage of cases the pulse, although it may be decreased in volume, is under 100, would bear out the above statement. In addition, the fact that there is no drastic drop in the blood pressure readings is also confirmatory evidence of the absence of shock features.

If temperature is present, it is almost always in the vicinity of 100 but in the earliest stages of the illness may be normal or even subnormal.

Tenderness, spasm and rigidity in the epigastrium are always present but not in any different manner as to their degree or localization that might give any aid to diagnosis. They are quite apt to be similar to that in perforated ulcer or in the colic of cholelithiasis.

IV—LABORATORY DATA

Leucocytosis is invariably present, averaging usually between 10,000 and 15,000 and by some it is thought that an extremely high leucocytosis, say in the vicinity of 25,000, is indicative that suppuration has occured or is occuring.

The blood sedimentation rate in these cases has been found to differ so erratically that it is not of any clinical use or value.

The urinary findings are negligible. There may or may not be an albuminuria of varying degree, bile may be present in urine, particularly in those cases in which some biliary pathological condition is co-existent, and sugar may also be present not due to the destruction of insular tissue but more often due to a pre-existing diabetes.

The blood sugar level is unreliable for use in determining anything of value as to diagnosis, prognosis or treatment. It was rather interesting to read a recent paper written upon this subject and finding the clinician doing glucose tolerance curves on an emergency patient, losing valuable time and subjecting a patient to a multitude of laboratory studies, which lead finally to a meaningless conclusion, while the patient probably passed on to meet his maker.

One procedure however, and one alone has been found to be of some help in the early diagnosis of acute pancreatitis and that is the determination of the blood diastase level. This point is of sufficient importance in a field so barren of help that further elaboration is not amiss.

First an explanation is in order as to the sources other than the pancreas from which amylase may be made available to the blood stream. These are the liver, the salivary glands and the duodenum. That these sources furnish an adequate supply of amylase is demonstrable by experiment. In complete extirpation of the pancreas in experimental animals there will be an initial drop in level with a subsequent return to a normal or slightly below normal level. It is this fact which explains why a blood diastase determination is of value only in the early phases of acute pancreatitis before a compensatory mechanism has begun to supply diastase from other sources.

The explanation for a rise in the blood diastase level in acute pancreatitis lies in the fact that with a rapid destruction of acinar membranes, such as takes place in this condition, there is a sudden overflow of pancreatic diastase into immediately surrounding pancreatic tissue not capable of holding this overflow or of confining it, so that there remains no other avenue of escape for it except into the blood stream. If coupled with this there is an obstruction of the pancreatic ducts, a vicious cycle is completed; there is more pancreatic tissue destruction, more ferment set loose and more taken up by the blood stream, so that an estimation of the amount present in the blood at such a stage will invariably show a very high amount over the normal.

The blood diastase level is, therefore, of help only in the earliest stages of acute pancreaatic disease, when the greatest amount of destruction of tissue takes place. If the process is an unusually severe one, death has intervened in thirty-six to forty-eight hours, and blood diastase determinations are meaningless. If the process is one which has reached its height at its very beginning or shortly thereafter, and the

destructive process is subsiding, the blood diastase level will be leveling off and will lose its significance. There is also the possibility in an extremely severe case, of the blood diastase level being below normal instead of above, because such an unusual amount of tissue destruction in the pancreas has taken place so quickly that no pancreatic ferment at all is being produced, none is available for absorption into the blood stream, and therefore, the blood content of it is low.

The value of repeated blood diastase estimation as an aid to judging whether the disease process is progressing or regressing is important. It should be done on an average we find of every four hours. Many cases have been cited in which the rise and fall of the diastase content of the blood exactly coincided with the peak and subsidence of the attack.

Serum lipase determinations follow about the same trend as serum diastase but the immediate clinical value of this test is sharply handicapped in that it requires a twenty-four incubation period before estimation can be done.

The clinical evaluation of the blood diastase test is perhaps best expressed in the words of Gray who summarized as follows, "elevated diastase values are always indicative of acute pancreatic disease provided the clinical picture resembling acute pancreatitis is present." Repeated normal blood diastase levels made early during the height of an attack of acute upper abdominal pain exclude the pancreas from consideration. In order that no smug complacency may develop with regard to this blood diastase test it would be well to enumerate some of its deficiencies: first, only a few laboratories in smaller hospitals and medical centers are equipped (although the equipment necessary is not extraordinary) to do quick emergency estimations of blood diastase. Second, there is a wide divergence of opinion as to what is the normal blood diastase level, as it varies widely in different individuals and under different conditions.

Low serum diastase levels, sudden drops or rises have not yet been adequately explained, at least to the point where one may have supreme confidence in using it as a diagnostic aid except in the one situation mentioned, early acute pancreatic disease.

Many clinicians prefer to use urinary amylase or diastase determination, believing that they have the same value as that of the blood. That is a matter of personal preference, but it might be mentioned that with all the divergances possible blood diastase levels fluctuate less widely than urinary. A lack of standardization exists with regard to both of these tests and should be remedied as soon as possible.

No better summation of the value of the amylase or diastase tests could be made than that of Gray quoted above.

The diastase value or normal human blood plasma ranges from 80 mg. to 150 mg. and the average about 115 mg. A negative finding in an acute upper abdominal emergency or rather a normal finding will definitely rule out the presence of acute pancreatic disease.

A laboratory technician should become familiar with the technique of doing the test by doing it at intervals on any type of case thereby decreasing his or her possibility and the establishment of a "normal" based upon the experience and technique of that individual laboratory.

The differential diagnosis of acute pancreatitis is by no means easy. Consideration must be given to any of the acute upper abdominal emergencies. Dr. Phil Thorek gave us eighty-four possibilities to think of in his paper. Most prominent in the order of the frequence of occurence will be acute cholecystitis, with impending gangrene or rupture, rupture of a gastric or duodenal ulcer or stone in the cystic or common duct. Less prominent but to be kept in mind are acute intestinal obstruction, mesenteric thrombosis, crisis of tabes, coronary thrombosis or occlusion.

It is in these cases in which keen perception in diagnosis and evaluation of minor details correctly can turn the scales and save a patient's life. If by a correct differential diagnosis acute pancreatitis is deemed present, the decision whether or not to submit the patient to operation arises.

Here several factors come into play, a good knowledge of which will aid in formulating a decision. Anatomically, the pancreas consists of multi-lobulated sections of tissue specifically differentiated to perform certain functions, mainly the production of digestive ferments. Each of these lobules is separated from the other by a connective tissue sheath, so that each lobule is a functioning gland all to itself and the whole is contained in a capsule, con-

posed of rather closely knitted connective tissue, not capable of much expansion. In acute pancreatitis the process is one of which necrosis involving many of these lobules or even all. A long incision into the pancreas from the head to the tail certainly cannot hope to accomplish much other than to release into the peritoneal cavity the highly toxic end products of a necrotic process and liberate tissue digesting ferments, particularly the proteolyte enzyme, trypsin. It is a question whether such a process would be of any help to the patient. It is obvious that to incise and drain each separate pancreatic lobule would not be feasible and ould accomplish no more than the above.

The removal of free fluid from the abdomen is about all that can be hoped to accomplish or simple insertion of a drain if suppuration or abscess is present, but is it not also the opinion of most that a closed peritoneum has greater defensive power than one which has been injured by incision?

In a patient already in a state of shock, his or her power of resistance definitely lowered, anesthesia and operation may constitute the added straw that he is unable to bear and be the primary cause of death.

It has been demonstrated repeatedly that the pancreas itself has remarkable recuperative power and that this power is enhanced in the presence of a closed peritoneum; therefore, everything should be done to avoid submitting these patients to operation if possible.

In those cases in which biliary change is a contributing factor, the problem becomes seriously complicated. Drainage of the gall bladder is the simplest surgical procedure feasible. Stone in the common duct, necessitating exploration of the duct, is usually too heavy a procedure to be borne well by a patient unless possessed of an unusual power of resistance and recuperation.

One can divide the cases of acute pancreatic disease into two types: the idiopathic acutely severe hemorrhagic and necrotic type, and the usually less severe but yet acute type in which there co-exists definite biliary tract disorders, probably the etiological cause of the disease.

It is here that keen diagnostic skill can come into play. If the symptoms are violent, the state of shock severe, the blood dastase and urinary diastase levels high, extreme conservatism and operative interference only as a last resort are indicated. Dr. Phil Thorek's dictum of carefully watching the patient's clinical chart comes into play here. If the symptoms are too violent, the state of shock not too severe, the blood diastase and urinary diastase levels sufficiently high as to be suggestive, a fair clinical judgment that a perforated ulcer or gangrenous gall bladder does not exist. Delay in operative interference is indicated.

If operation is decided upon, it should consist only of drainage of the gall bladder and the suction and removal of any fluid in the peritoneal cavity.

An abrupt reversal in the management of these cases has taken place. Formerly, the dictum was that the earlier the operative intervention the better were the patient's chances, this in spite of the fact that operation per se accomplished nothing more than a "look see," a verification of diagnosis at best, and injured the invasional defenses of an intact peritoneum. As we look back in retrospect it is hardly probable that the mere suctioning off of some hemorrhagic fluid was of any help to the patient. Those who did survive operation did so by the Grace of God and a strong constitution, and not because of any surgery. Operation is indicated where there is any doubt as to the diagnosis rather than let a patient die from a possible ruptured ulcer.

Today that is changed and for the better. A study of comparative statistics of ten, twenty and thirty years ago shows that diagnostic acumen has undoubtedly improved if measured by the higher percentage of accurate diagnosis, and the mortality tables lowered as measured by the number of cases listed as recovered.

The accepted treatment today is supportive and symptomatic in all cases except those in which the indications are that suppuration has intervened. Then and only then surgical drainage might be of benefit. Expectant therapy, therefore, divides itself into the following measures:

1. Relief of Pain by Liberal Use of Morphine. In addition to the use of morphine personal experience in the use of nitroglycerine has been gratifying although in too limited a number of cases. Dr. Phil Thorek has just finished telling you about his own very favorable experience with nitroglycerine in such cases where edema and spasm might be contributing factors to pain. A number of French writers have been using ephedrine and their reports are enthusiastic. I

personally have not as yet had the opportunity to use ephedrine in any case and therefore can offer no opinion corroboratory or otherwise.

Theoretically, one may claim advantages for its use; it has anti-spasmodic and vasodilation properties. Through use of these it is conceivable that should any spasm of the sphincter of Oddi be present it could be overcome. In its action of dilation of the common bile duct one would be warranted in assuming that the smaller ducts such as the duct of Wirsusg or Santorini might in some degree also be dilated. Its use would tend to overcome any increase in biliary pressure or any reflux of bile due to such pressure, were it present. The above is along the same line of reasoning as that suggested for its use as an aid in the passage of a stone left behind in the common duct at operation.

2. Relief of Gastric Distress. First and most mportant here is that nothing shall be given by mouth, as stimulation of pancreatic ferment production in the tissue still intact would take place and thereby hinder rather than help the situation.

3. An indwelling Levin catheter with the frequent removal of stomach secretions is of material help. It is not necessary to have continuous suction, such as in cases of obstruction.

4. Maintenance of Fluid Balance. Here the question of administration of glucose is a case of impaired pancreatic function brings up a problem. First, the question of stimulation of pancreatic activity, which is to be avoided if possible, and secondly as both hyper and hypoglycemia states have been found in this condition, the use of intravenous glucose is debatable. In fact, in some cases in which shock has been present it was debatable whether the state of shock was due to the disease or to a hypoglycemia present as the result of the overproduction of insulin. One may take a middle of the road path by giving with glucose solution a sufficient amount of insulin to counteract it. Physiological saline or Ringer's solution may be used in amounts corresponding roughly to an estimation of the fluid loss caused by vomiting and perspiration.

Surgical intervention is indicated wherever there is reasonable doubt as to the correctness of the diagnosis or whenever signs of suppuration intervene. Dr. Phil Thorek's dictum of carefully watching the pulse rate plus leukocyte comes into play here too.

In advanced pancreatic necrosis or in pancreatic abscess, drainage of the pancreas is accomplished by incision of its peritonea capsule and the insertion of multiple drains into either definite necrotic or suppurative areas.

The care of the skin and abdominal wall against the action of the tryptic ferments released by drainage is distressing to say the least and is worthy of a written paper of itself.

Some have advocated cholecystectomy as a measure for relieving biliary compression. That is a matter of surgical judgment based upon the individual case but it is difficult to see where it presents many advantages.

It is a more prolonged operation superimposed upon a patient who is already depleted in physical reserve because of his fight against an already present serious condition, and it is difficult to visualize wherein removal of the gall bladder will be in sufficient time mechanically to help relieve an edematous duodenum which is acting by the edema present to close the sphincter of Oddi. Bile will continue to come down the hepatic duct and if there is an impassable barrier at the duodenal and bile duct junctions the same conditions for increased tension and reflux of bile into the pancreatic duct, although it is admitted in perhaps a slightly lesser degree, will remain.

It would seem that cholecystostomy offered more advantages. It is faster and easier to do and carries less shock to the patient. It decompresses the biliary system immediately by offering another outlet for the escapeof bile. If gallstones are present in the gall bladder, they can be removed. There is one prime factor which must be present when deciding to do a cholecystostomy, and that is the cystic duct must be patent. It is quite obvious that draining he gall bladder above a closed or blocked cystic duct would hardly help in decompressing a hypertensive biliary system.

Those against cholecystostomy argue that there is a high rate of recurrent pancreatitis following this operation which does not exist after cholecystectomy, and that cholecystectomy has to be done eventually for relief of this recurrent pancreatitis, so why not do it in the first place?

A careful and fairly exhaustive review of the litetrature of the past forty years is rather barren of citations of cases wherein acute pancreatitis occured a second distinct and separ-

ate time after survival of a first attack. Exacerbations of the primary attack, yes, but a second siege separated by a reasonable interval from the first one is very rare.

A word might be said as to the prophylactic removal of the gall bladder in each and every case in which stone is present and in those cases of chronic cholecystic disease of dysfunction without stone which do not respond satisfactorily to adequate medical management as a possible preventive of later pancreatitis. So much evidence exists of the high association of gall bladder disease with pancreatitis that much weight could be given to the proposition that it might be one of the more important etiological factors in its production.

Incision and drainage of the common bile duct is recommended by others and presents many advantages from the standpoint of relief of biliary pressure, but it also presents disadvantages in that it is a more extensive operation to impose upon an already seriously ill patient.

Primarily, surgical intervention in these cases is unwarranted unless signs of biliary tract obstruction co-exist or evidence of suppuration supervenes. Operative risk progressively increases after the first twenty-four hours.

The treating of these patients conservatively has resulted in a marked diminution in mortality. This can be seen by comparing the varying statistics available.

CONCLUSIONS

1. The most important item in the entire field of pancreatic disease is that in deciding for or against operation in the acute cases, to judge whether only an acute edema of the pancreas is present or if necrosis or suppuration are present whenever this is possible.

2. There should be a wider use of blood diatase determination with a full appreciation that only in early acute cases can its possibilities be utilized.

3. The use of ephedrine in the early case where it can be assumed that edema alone is present, that more comprehensive studies and data be made available is recommended. The encouraging but meager reports available justify such a plea.

4. The prophylactic removal of a diseased or poorly functioning gall bladder which fails to respond to adequate medical therapy is shown by experience to be of value. The same removal of a gall bladder containing stone goes without saying. As a prophylactic or preventive measure against acute pancreatitis, it is definitely recommended.

5. Statistical evidence favors conservatism in the handling of the acute cases and the mortality is much lower whenever this has been practiced.

MODERN MANAGEMENT OF OEDEMA

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OVER TWO thousand years ago Hippocrates taught his students that disease is a disorder of body fluids. Many other theories have held the attention of physicians since that time but careful observers of medical progress will note that today more and more attention is being devoted to these long neglected fluid or humoral relationships in the body. Oedema is but the end result of a profound disturbance in the salt and water metabolism of the body. As such, it is of importance not only to cardiologists, but to students of all diseases in any one of which the subtlest change in fluids and electrolytes may have the most far reaching significance.

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We are not here concerned with the various theories of oedema formation, however interesting they may be, but rather with the therapeutic problem of clearing oedema; although this may depend in large measure upon the specific aetiology of the oedema, the fluid produced by different mechanisms is of such striking uniformity of composition that the same principles may be applied in ridding the body of oedemas of different origins.

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Oedema is a simple increase in the interstitial fluid of the body. Such fluid can accumulate and remain only if its constituents are supplied and remain. Just as a patient cannot put on weight unless the caloric intake exceeds the energy expenditure, so oedema can arise only if the intake of the elements composing it ex-

ceeds the output. It has been shown that patients who have had congestive heart failure are more susceptible to the production of oedema upon the ingestion of salt than are controls[1]. It also seems unlikely that the kidneys of patients with congestive heart failure handle salt in any manner qualitatively different from that of normal persons[2]. Probably, circulatory disturbances in the kidney prevent it from excreting as much salt as does the kidney of the normal person and the salt is therefore deviated to the interstitial spaces. During the onset of congestive failure the patient is in what may be described as "positive salt and water balance."

Oedema fluid does not differ markedly from normal extracellular fluid or from plasma. It consists of an alkaline solution composed chiefly of sodium salts, principally chlorides and bicarbonates. The chloride and bicarbonate are in the ratio of approximately five to one. The total salt content of the fluid is approximately 7.5 Gm. per litre of fluid. Oedema fluid contains more chloride and potassium than blood serum whereas the quantities of sodium, bicarbonate, calcium, urea, glucose, and nonprotein nitrogen are about the same and the protein content is usually much smaller than that of serum ([3, 4, 5, 6]). The composition of oedema fluid is subject to the same variations as is the normal extracellular fluid and different degrees of concentration of the salts relative to the salt concentration of the neighboring cells may exist. The fluid is more often than not hypertonic for the surrounding cells. Consequently, patients with oedema are not so much water-logged as "brine-logged." Even the most casual observer of patients showing congestive heart failure with oedema must notice the extreme thirst and dry tongue of which they frequently complain. Such patients have what is actually true dehydration, that is, a plain water deficiency. Unfortunately, the word *dehydration* is loosely applied to salt and water deficit with loss of extracellular fluid volume such as is found in burns and shock.

The body tends to hold all its substances in steady concentration and sacrifices volume before changing concentration. It is therefore not possible to dispose of oedema until the salt as well as the water of this fluid has been eliminated. This explains the surprising ineffectiveness of mere water restriction without salt restriction in ridding the body of oedema[7].

A certain amount of fixed base is excreted in the urine when the kidneys eliminate metbolic and ingested acids. The kidneys are able to adapt the excretion of this base to the particular exigencies of the moment. It has been well said that the kidneys are the ultimate guardians of the internal environment. They perform this function well and if given adequate water they can control a tendency either to acidosis or to alkalosis by altering the constituents of the salts they excrete[8, 9, 10]. If there is a tendency to acidosis, sodium is withdrawn from the intercellular fluid to neutralize the acids and the kidney excretes the resultant neutral salts. Such a situation is encountered in certain forms of renal disease in which acidosis occurs due to retention of acid radicles. This mechanism is used in the treatment of oedema, when, by inducing a tendency to acidosis, we seek to mobilize the sodium of the intercellular fluid and have it excreted through the kidneys. After the salts of the oedema fluid have been thus eliminated, the water of the fluid is free either to be excreted through the kidneys, skin or lungs or to replenish the water of the dehydrated cells.

The sodium used to neutralize the acids, whether metabolic or ingested, leaves the body in solution as urine. But water can reach the kidneys only after the other requirements of the body for water have been satisfied. The kidney is not concerned with producing ideal urine but with maintaining ideal blood and body fluids. Urine water is merely what the body can spare from its primary requirements. The amount of water reaching the kidney will therefore depend upon the more pressing demands of other bodily needs. Patients who have fevers or who are perspiring profusely in hot weather will divert much water from the kidneys to the sweat glands for temperature regulation. Patients who are dehydrated will likewise divert water to replace the reservoir of their intercellular fluid before allowing much for the kidneys. This situation can occur in the presence of oedema; if the oedema fluid is hypertonic for surrounding cells when the salts of the oedema fluid are eliminated, some of the freed water will be withdrawn into the cells and will not be available for the manufacture of urine. A third reason for supplying abundant water to the body for the formation of urine is provided by the impaired functional state of the kidneys of most patients with oedema. A kid

ney with low concentrating power naturally needs much more water to excrete a given amount of sodium than one with high powers of concentration[11].

The objects of a regime designed to rid the body of oedema are therefore threefold. In the first place, since the kidneys are unable to handle salt as effectively as they should, their load should be reduced and this can be done by the restriction of sodium in the diet. Thus, diversion of unexcreted salt to the intercellular fluid with consequent formation of oedema can be greatly diminished. Secondly, the tendency of the metabolic acids of the body to neutralize the sodium of oedema fluid can be encouraged if the natural leaning of the body to acidosis is increased through dietary regulation and the supplementary use of acids and acid salts. Thirdly, the elimination of sodium through the kidneys can be facilitated and true cellular dehydration prevented by allowing the patient an abundance of water.

The first requisite in the management of these patients is a diet low in salt and sodium. The average person normally ingests between 5 and 12 Gm. of salt daily according to his liberality with the salt shaker. The usual hospital "low-salt" or "salt-free" diet actually contains often as much as 3 to 4 Gm. of salt a day and is not much improvement over simple removal of salt added from the table. The daily diet of these patients should contain between 1 and 2 Gm. of salt. The original Karrel diet is low in salt by virtue of the fact that it is low in everythng and is in effect a starvation regime. It contains about 1 Gm. of salt daily in 800 c. c. of milk divided into four portions. Such a diet is suitable for the more severely ill patients in the acute stages, but if continued long, will soon cease to satisfy both the appetite and the dietary requirements of the patient. Other more varied diets must be used. It has been found infeasible to reduce the salt in the diet below 1 Gm. daily. If diets containing as little as 0.5 Gm. are used, although the oedema may subside some what sooner, the protein intake is inadequate[7]. This is not surprising when we consider that 45 Gm. of animal protein contains 1 Gm. of salt. It must be remembered that the reduction of salt alone will not do much towards clearing oedema. Every gram of salt holds with it in the interstitial fluid 150 c. c. of water. Reducing the salt intake from 5 to 1.5 or 2 Gm. daily will therefore provide for a diuresis of only about 500 c. c. However, the reduction is important in the prevention of further oedema.

According to Schemm[12] it is more important that the diet have a neutral or acid ash than that it be low in salt. By this means the diet adds no new excess basic elements to the body and the naturally produced metabolic acids are neutralized by intercellular sodium which is withdrawn from the oedema fluid. Oedema has failed to clear in some cases when the salt content of the diet was reduced to 0.5 Gm. daily[7], and the explanation may be that the diets, even though low in salt, were basic in reaction. Acid ash diets suitable for use in these patients have been published by Newburgh[13] and have recently been simplified to the point of practicality by Schemm[12]. Such diets are built simply upon the fact that milk, all vegetables and all fruits except prunes, plums and cranberries yield an alkaline ash. These foods are therefore restricted but an abundance of acid-ash foods such as meat, chicken, eggs, fish, cereal foods, brown bread and the three excepted fruits is allowed. The diets are, in addition, low in salt and sodium, and contain about 0.85 Gm. sodium and 2 Gm. salt. They may be easily adjusted to accommodate other diseases such as obesity, peptic ulcer and diabetes. They are no more complicated than diabetic diets and are probably even less trouble to prepare. Although they may require iron and vitamin supplements, this can be easily arranged. Unsalted butter can usually be bought from a dairy and unsalted bread, if not obtainable from a bakery, can be made by using water (instead of milk) and salt free butter without much trouble or sacrifice of taste. The patient may, however, complain of boredom with the unsalted food but the tastelessness can usually be alleviated considerably by allowing the addition of granulated ammonium chloride used in a salt-shaker. The only objection to the similar use of potassium chloride is its stinging taste.

The acidifying tendency of the diet is usually enhanced by the administration of acidifying agents such as ammonium chloride and sometimes dilute hydrochloric acid. These substances probably act as diuretics by withdrawing fixed base from the extracellular fluid for the neutralization of their acid radicles. It is also conceivable that the acidosis they induce alters the osmotic pressure forces of the extra-

cellular fluid in accordance with the Donnan equilibrium theory and that a consequent withdrawal of body water (and associated electrolytes) results[14]. If ammonium chloride is ingested, the ammonium radicle is for the most part changed to urea in the liver and the urea content of the blood and urine rises. Only slight, if any, increases in ammonia in the systemic blood are detectable[15]. Removal of the ammonium radicle then permits the development of acidosis secondary to preponderance of the chloride radicle. The blood chloride rises and the carbon dioxide combining power decreases. This latter effect is obtained with doses of over 5 Gm. daily; if the doses exceed 8 Gm. daily, there is a definite lowering of the blood pH. The kidneys compensate for the increased acid by an increased production of ammonia. The compensation is, however, incomplete and a certain amount of fixed base is necessarily withdrawn from the body along with the excess acid radicles. This amount will naturally be greater in patients with renal disease in whom the power of ammonia formation is impaired. In a case of Gamble[14] about 50% of acid radicle remaining after formation of urea was covered by the new ammonia formation and there was in this patient (with renal disease and oedema) a loss of considerable fixed base. Haldane[16] in experiments on himself found on ingesting ammonium chloride that the amount of ammonium chloride appearing in his urine was about two-thirds of the amount ingested. In his case, therefore, with presumably normal kidneys, one-third of the acid salt ingested was available for the removal of fixed base from the extracellular fluid. If we accept this figure, then, the ingestion of 6 Gm. of ammonium chloride daily would give about 2 Gm. of excess acid radicle available for the withdrawal of fixed base. Less than 2 Gm. of sodium would be withdrawn and with this amount would go not more than 600 c. c. of water. This is not much in a severely congested patient and even this figure would not be attained if the smaller amount of 4 Gm. daily advocated by Schemm[12, 17] is given. Therefore, the drug might as well be offered in more effective quantities and in the experience of the author better results have been obtained by giving 5 to 7 Gm. daily. However, ammonium chloride is not to be prescribed flippantly or indefinitely. If large amounts are used, severe acidosis may develop and short of this there may be compensatory hyperpnoea which may increase the respiratory discomfort of the patient. The work of the kidneys is increased by the excretion of the extra solids and the functions of the liver may be depressed. Plain coated tablets of ammonium chloride may cause nausea and even vomiting from gastritis, and enteric coated ones, although more palatable, may not always dissolve.

It is probably better to give ammonium chloride cyclically and if mercurial diuretics are being used, it is better to synchronize the two medicines so that the ammonium chloride "prepares" the patient for the mercurial. The action of the mercurial diuretic depends upon the depression of tubular reabsorption of chloride. The use of ammonium chloride increases the chlorides available for this "non-reabsorption." When plasma and tissue chlorides have become depleted as a result of combined salt restriction and mercurial diuresis, the mercurials cease to have any effect until chlorides are replenished and this can be done best by the use of ammonium chloride. However, this depletion can be prevented and the mercurial action enhanced if the ammonium chloride is given regularly for three or four days before the injection is administered.

Dilute hydrochloric acid is an excellent adjuvant to the action of the ammonium chloride. The water drunk by the patient can be acidified by the addition of 0.5 to 0.7 c. c. of dilute (10%) hydrochloric acid for each 200 c. c. of water. In this way about 5 to 10 c. c. of dilute hydrochloric acid may be taken easily and if the patient does not object to the taste, much larger amounts may be given. If 10 c.c. are taken, the patient ingests the equivalent of about 1.4 Gm. of ammonium chloride which may be of distinct help. Such acidified water is best taken through a straw.

The final requisite is to ensure an adequate intake of water. It is generally agreed that every person needs 1500 c. c. of water daily for water of vaporization and stool and 1500 c. c. for satisfactory urine volume[11]. Some water, perhaps as much as 1000 c. c., is naturally derived from the food in the diet. Apart from this, however, at least 2500 to 3000 c. c. of water is required daily by the average patient if his normal needs are to be satisfied. As already mentioned, the necessity for water is greatly increased if much water is used for temperature

regulation or if the kidneys are impaired. The development of oedema does not free a patient from these varied demands and his fluid intake should be high enough to gratify them. It will need to be even higher if he is dehydrated and this may well be the case despite the presence of oedema. Workers in this field are becoming increasingly aware that fluids should be allowed freely to patients with congestive heart failure[7, 19, 20].

Schemm[12, 17, 21] has gone even further and believes that a high fluid intake, so far from being detrimental, is actually beneficial to such patients. Although Schemm in his latest article[21] denies that any of this effect is due to "washing out" of salt, there is considerable evidence to the contrary. Stewart and Rourke[22] showed that administration of considerable amounts of glucose solutions caused reduction of the volume of extracellular fluid as indicated by loss of weight and sodium. More recently, Wolf[23, 24] has shown that if water is administered continuously over long periods, the urine output exceeds the fluid intake and dehydration results. In his experiments, when a steady state had been reached the urinary output alone was 8 per cent more than the intake rate and the actual loss of water must have been greater becarse skin evaporation was going on at the same time. These results appear to be due to the fact that for every cubic centimetre of urine there is a minimal diuretic excretion of 1.2 mgm. of chloride and if water only is given, there is a consequent loss of body chloride. Additional water is lost to maintain a constant concentration of chloride. Therefore, the more urine is formed, the more will chloride be lost and oedema cleared.

The present author has found such high-fluid intakes often extremely effective and is quite uninhibited in the use of relatively large quantities. As much as 8000 c. c. of water in a single day has been given to a severely congested patient with marked benefit, and Schemm has often used such amounts. Occasionally, a patient may complain of a sense of fulness and the oedema may actually increase for a day or so as the tonicity of the oedema fluid is reduced by the water, but these symptoms are transitory and insignificant. In two of his patients Schroeder[7] found that sudden increases in fluid intake produced symptoms of water intoxication, presumably due to dilution of electrolytes. These patients had severely damaged kidneys which were apparently unable to take care of the new load of water and there was diversion to the intercellular spaces. The occurrence of the symptoms noted in Schroeder's two patients who had weakness, prostration, muscular cramps and drowsiness is, however, very rare in these cases. They have not been encountered by the present author or by Schemm[17], and they are in any case not serious. They are mentioned to stress the point that indiscriminate use of any therapeutic measure may be detrimental and there may be some patients in whom a high fluid intake is not feasible or beneficial although it is believed that such cases are extremely few. After oedema has cleared, the water intake should naturally be reduced to the amount necessary to satisfy the normal needs of the patient, taking into consideration the previously mentioned factors of temperature regulation and kidney function. High fluid intakes after oedema has cleared may lead to dehydration as indicated by the work of Wolf[23]; furthermore, large amounts of water in surgical patients who are not oedematous may apparently cause derangement of renal control of plasma electrolyte composition with depletion of sodium[22], a situation which will not occur in these patients if the water intake is reduced as the oedema clears.

Lest it be argued that such a high fluid intake will over-burden the heart, it may be stated (quoting Schemm[12]) that an oral intake of 8000 c. c. daily adds only 16 pounds to the four tons of fluid moved by the heart in half a day. If one litre of water is given by vein in an hour, it moves along with the 150 to 300 litres ordinarily moved by the heart in that time. The circulation is protected from overburdening by the valves present in the ability of the kidneys to excrete water rapidly and by the ability of 68,000 square feet of capillary bed to dilate and develop a sieve-like permeability to water and electrolytes.

The actual amounts of water given in excess of the natural bodily needs will vary with individual circumstances. There is little merit in giving large amounts of water when oedema is responding to other measures; on the other hand, the present writer has noted (as did Schroeder[7] and Schemm[17] that increasing the fluid intake adequately may produce improvement in previously unresponsive patients. If

the fluid cannot be taken by mouth, it can be given intravenously and is best given as a solution of 5% glucose. A 10% solution of glucose will only add to the dehydration (through glycosuria without supplying water to the cells.

This regime in no way conflicts with other measures being undertaken for the treatment of the oedema itself or the primary cause of the latter; it is certainly not intended to replace measures directed toward improving the circulation. The diuretics of the xanthine and mercurial groups may be freely given and in the more advanced cases are usually necessary. (It is, of course, all the more important to supply abundant water when a strong mercurial is being administered if dehydration is to be prevented). It should never be forgotten that in heart disease digitalis is still the most important of all diuretics; indeed, it was the diuretic effect in dropsy which was first noticed and which led Withering to introduce it into medical practice long before its significant actions were fully appreciated. Although Withering was well aware of the effect of digitalis on the pulse and the heart, he was principally impressed by its almost incredble diuretic effects.

Although the regime outlined is simple, it nevertheless will fail unless due attention is paid to detail. From the beginning intake and output must be carefully measured and it is even more important that if possible the patient be weighed daily before breakfast. Since an increase of 8% of the weight of a limb is necessary before pitting oedema appears, changes in weight are obviously much earlier indications of changes in progress. The patient must be cautioned to follow the regime carefully at first; later, relaxations may be permitted according to circumstances. However, constant vigilance is necessary to avoid apparently trivial but perhaps significant changes in the diets and other features of the regime. Care must be taken to see that no added seasoning or salt and no saline cartharties or soda for indigestion are taken. Schemm[21] has recently pointed out some of the commoner pitfalls in this regime. The physician must devote some time to the explanation of the elementary theory and practice of the regime; for if the patient's interest can be stimulated and later gratified by practical results, he will usually follow the program faithfully.

Yet it must not be thought that this treatment can materially affect the primary condition producing the tendency to oedema except in so far as the nutrition of the tissues (including those of the heart) is improved and the burden of the heart decreased by removal of the oedema. The symptomatic relief, however, is usually profound, and alone would justify the measures adopted.

Several illustrative cases are briefly cited.* Case 1: W. M., a 50 year old man who had bronchial asthma and chronic bronchitis with pulmonary heart disease (chronic cor pulmonale) and arteriosclerotic heart disease, manifested marked congestive heart failure with anasarca which failed to respond to a previous regime of digitalis and bed rest. Fluids were forced abundantly so that for the eighteen days of his hospital stay he never took less than 3500 c.c. of water daily, and averaged 5000 c.c. daily, attaining on one day an intake of over 8000 c.c. His urinary output rose from 1500 c.c. the first two days to 7000 c.c. on the fifth day and for the next thirteen days was always over 3000 c.c. daily. His weight dropped during this admission from 168 to 140 lbs. and he was discharged with a residual cough but without any evidence of congestion. This patient might, of course, have done very well on a low fluid regime but the case at least illustrates the fact that no harm need come from forcing fluids in severely congested patients.

Case 2: E.M., a 52 year old woman with hypertensive and arteriosclerotic heart disease and marked congestive failure, had previously failed to respond to a variety of measures; on the regime outlined in this paper her weight dropped from 170 to 140 lbs. in ten days. Her fluid intake was not as high as in the previous case due to lack of cooperation, but it was never less than 1800 c.c. daily and usually well over 2000-2500 c.c. daily.

Case 3: J.O'C., a 78 year old man who had arteriosclerotic heart disease and auricular fibrillation with a slow ventricular rate, manifested severe anasarca on admission. After thirteen days of the treatment described in this paper his weight had dropped from 132 to 110 lbs. The important point about this patient is that the ventricular rate was originally slow (without digitalis), being around 70 to 80 and rarely above. He was not digitalized and his heart rate did not decrease to any significant degree when he was put to bed. The changes in the extracellular fluid and the loss of oedema may

therefore reasonably be attributed to the regime prescribed and hardly at all to changes in circulatory dynamics. He was started on the Karrel diet and later given a low-salt acid ash diet. Despite poor cooperation in drinking water his intake was usually around 2000 c.c. daily.

Case 4: S.M., a 3½ year old boy, had chronic glomerulonephritis and considerable oedema which had always increased with upper respiratory infections and had been extremely intractable. Upon admission the boy's weight was 45 lbs. and he was very oedematous. He was kept on an ordinary salt-free regime for some days without change. He was then put on a low-salt acid ash diet, fluids were forced, his water was acidified and he was given large amounts of ammonium chloride. His weight dropped in nine days to 34 lbs. It was mantained there for a few days and he was then taken off this regime and placed again on the former one. His weight began to increase and within another nine days (from the second change of regimes) it had reached 42 lbs. again. At this point he was once more very oedematous and he was replaced on the high fluid, acidifying regime and once more his weight dropped within eight days to 34 lbs. This child was discharged completely free of oedema and in other respects remarkably well. He continued on the regime at home with some relaxations and got along very satisfactorily for about five months. Then, he had another respiratory infection and returned with further oedema. He was given penicillin and the regime for oedema was applied aggressively with a satisfactory and rapid response. This case is instructive not because the child responded to this regime better than to others, but because he served as his own control; the case also illustrates the applicability of the treatment to oedema of other than cardiac origin.

Case 5: W.B., a 56 year old man was admitted to the hospital with hypertensive cardiovascular disease, marked congestive failure and some bronchial asthma. He had severe hypertension, bundle-branch block, gallop rhythm and pulsus alternans, every indication, in fact, of an early termination to his long illness. He had responded poorly to the usual measures employed in such cases and mercurial diuretics, even when given frequently, did not control the oedema satisfactorily. The regime outlined in this paper was then applied vigorously. On the first day his intake was increased from the 1100 c.c. of the previous day to 5200 c.c. His output that day was only 1800 c.c. which was barely double the 900 c.c. he had eliminated the day before. The oedema had obviously increased and the patient's symptoms of congestion such as dyspnoea, not to mention his anxiety, had increased. He was encouraged, however, to continue drinking and his intake for the next day was about the same, being 5100 c.c. To relieve the disquietude of the patient (and some of the doubts of the physician) he was given a mercurial diuretic that morning. His output that day was no less than 13,000 c.c. and no one was more astonished or pleased than the patient himself. His weight, which had previously remained on a plateau of 152 lbs. for several days, dropped in that one day to 132 lbs. The effect could not be attributed solely to the mercurial, although this may be said to have precipitated the diuresis, because he had previously received mercurial injections without nearly this response. As a matter of fact, after this diluvian result the patient lost no further weight, but he continued to drink water and his output was maintained at a satisfactory level. Within a short time he had improved enough to be discharged. There is no implication here that the eventual prognosis has been altered, but the patient was undoubtedly made more comfortable. The case aply illustrates the enormous exchange of fluid which can take place in congested patients not only without harm but with apparent benefit. No reaction from the tremendous diuresis was seen because the patient was receiving adequate amounts of chloride and water and thus the usual toxic effects of marked mercurial diuresis due to hypochloraemia and dehydration were obviated.

It will be seen that the principal recent change in the approach to oedema is relaxation of the habitual restriction of water for these patients. Yet it is not generally known that earlier clinicians not only permitted a high water intake for patients with congestion but encouraged it. William Withering wrote in his original account of the foxglove, "I allow, and indeed enjoin my patients to drink very plentifully of small liquors through the whole course of the cure; . . . The patient should be enjoined to drink very freely during its operation. I mean, they should drink whatever they prefer, and in as great quantity as their appetite for drink demands. This direction is the more necessary, as they are

very generally prepossessed with an idea of drying up a dropsy, by abstinence from liquids, and fear to add to the disease, by indulging in their inclination to drink.''[25] The attitude of Withering on this matter was also that of nearly all other physicians until as recently as fifty years ago. Unfortunately, the practical observations made by the earlier clinicians were then jostled aside by an accumulation of fascinating concepts opposed to the idea of allowing a free fluid intake in oedema. For many years the unthinking authority of tradition has restricted fluids in such patients, aptly illustrating the belief of Sir Francis Bacon that the greatest enemy of truth is authority. It has only been through the work of such investigators as Newburgh and Coller and the the exhaustive clinical applications of Schemm that there has once more been assembled enough evidence from practical experience to displace these fancy but irrelevant theories.

APPENDIX: Diet sheet supplied to average (uncomplicated) patient, largely reprinted from Schemm.[12]

SCHEMM DIET

The purpose of this regime is to maintain the circulation at its greatest efficiency and to decrease the tendency of the body to retain fluid. The latter results from the abnormal retention of sodium. If the sodium can be adequately eliminated from the body, the amount of water drunk will not affect the fluid held by the body and in fact the more water drunk the better. The sodium is eliminated by arranging the diet and medicines so that there is always a slight preponderance of acid which the sodium is required to neutralize. It is more important for this slight acid to be present than it is for the diet to be particularly low in salt although the latter is also desirable.

DIET: The diet is based on the fact that milk, all vegetables, and all fruits (except prunes, plums and cranberries) yield an excess of alkaline ash and these should therefore be avoided in favor of opposing foods such as meat, chicken, fish, eggs, cereal foods, and the three excepted fruits mentioned above. In addition table salt and other sodium salts should be restriced to the lowest possible point.

Full Neutral Diet (Low-Sodium, Low Salt. Acid-ash. Calories unrestricted).

FOODS UNRESTRICTED AS TO AMOUNT (from which at least two or three servings must be taken for any one meal):

EGGS: Two equal one serving (which can be substituted for a meat serving).

MEATS: Meat, fish, or chicken, one serving of about ¼ lb. per day.

BREAD: Plain breads without nuts or raisins. Whole wheat bread is preferable. Five slices or cereal food servings as substitutes in each day.

CEREALS: The following only (one serving a day at least) — oatmeal, farina, quickcooking cream of wheat, cracked or ground whole wheat, corn-meal mush, hominy, puffed rice or puffed wheat, muffets.

CEREAL FOODS: Servings may be taken at any meal and must be taken if meat or egg is not eaten: Macaroni, spaghetti, rice, homemade noodles, corn.

FRUIT: Prunes, plums, or cranberries. (Other fruit listed below).

FOOD RESTRICTED AS TO AMOUNTS (from which NO MORE THAN TWO servings should be taken at any one meal):

VEGETABLES: Two servings a day of ½ cup each of any vegetable except parsnips, lima beans, rhubarb, chard, and spinach, which are forbidden. One small potato equals a serving. Use fresh or frozen vegetables or those canned without salt.

FRUIT: One serving of ½ cup of fruit juice daily except raisins and dates which are forbidden. Salads of fruits or vegetables may be made from the above, as desired. Raw fruit and raw vegetables should be used several times a week.

MILK AND MILK PRODUCTS: Two cups of milk daily, including that used in preparing the food.

CREAM: Two tablespoonfuls in coffee or tea, ¼ cup for breakfast cereal.

ICE CREAM: without fruit or nuts, one small scoop in a day.

CHEESE: Only unsalted cottage cheese which may be substituted for a meat or egg serving.

OTHER FOODS AND FOOD COMBINATIONS:

SOUPS: May combine vegetables, as allowed above, with the milk allowance or with salt-free broth to make soups. Salt-free clear beef or chicken broth may be taken as desired both with and between meals.

DESSERTS: No limits as to amount: plain

jello, wine jelly, plain tapioca, angel food or sunshine cake. (No cake or cookies made with salt, soda, or baking powder.

Limited by milk allowance above: Custard, junket, cornstarch pudding, eggnogs, ice cream. Fruit as indicated above.

BEVERAGES: One cup of tea or coffee to each meal, chocolate made with part of milk allowance.

NEUTRAL FOODS: Unrestricted: Sugar, butter, oil, gelatin, salt-free salad dressing, plain tapioca or plain cornstarch, clear sugar candies. Butter must be salt-free.

SAMPLE MENU

BREAKFAST:

½ cup orange juice	¼ cup cream
1 soft boiled egg	1-2 slices toast
and/or cereal	coffee

LUNCH:

Corn soup	Lettuce salad
1 poached egg	Bread Milk, 1 cup
on toast, or buttered noodles	½ cup baked custard

SUPPER:

Roast beef	Bread
1 small baked potato	Coffee
½ cup asparagus	½ cup plums

PRECAUTIONS FOR DIET

1. No salt or soda to be used in the cooking or at the table. If a salt substitute is required use small amounts of ammonium chloride. Use no other salt substitutes.

2. Obtain unsalted sweet butter or wash butter free from salt. Obtain unsalted bread from baker or make at home. Unsalted salad dressing must be made at home.

3. Take no salted appetizers or salted foods such as nuts, potato chips, sardines, olives, pickles, relishes; no cheese except unsalted cottage cheese; no smoked or salted meats or fish such as canned salmon or tuna, bacon (unless parboiled), ham, lunch meats, sausage, or salt pork.

4. For "gas" or indigestion — take no bicarbonate of soda or other alkali powders or tablets — use calcium carbonate only.

Avoid cabbage family — turnips, rutabagas, peppers, radishes, onions, spices, greasy fried foods, and pork.

5. For extra liquids — Take none of the vegetable juices or fruit juices on the restricted list, or milk or salted bouillon. Use only well-diluted plum, prune, or cranberry juice or water with fruit flavoring or unsalted chicken or beef broth.

6. If less of the acid ash foods is eaten for some reason, less of the basic ash foods should also be eaten. Whenever possible, however, a full mixed diet of the allowed indicated portions should be taken.

* Cases admitted to St. Joseph's Hospial, Phoenix, Arizona.

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THROMBOCYTOPENIA WITH SPLENECTOMY
(Case Report)

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Frank J. Milloy, M. D.

THIS twenty year old, white, American college girl was first seen on October 7—twelve days before entrance to the hospital. The chief complaint at that time was continuance of the menstrual flow which began six weeks previous. Aside from this condition, she felt perfectly well, had an excellent appetite, but remained in bed due to the continuous menstrual flow. Concerning her past history, she had had the usual childhood diseases and, in addition, was subject to asthma, although she had experienced no severe paroxysms. About six months prior to the time when first seen she had some bluish, discolored spots on her body, and consulted a physician who made some blood tests and told her that the platelet count was low. She went away to college in the fall.

When the present menstrual flow began, she was examined by another physician who found the platelet count low again. She was placed in liver, thiamin chloride and synkamin by mouth. When the flow persisted she was sent home. Now she was placed on enteric-coated stilbestrol 1 mg. daily by mouth. She was seen again one week later. During this time the menses had continued unabated. She still felt very well but had developed hemorrhagic spots on the mucous membranes of the mouth and a few small areas on the body. The picture seemed to be quite typical of purpura hemorrhagica. At this time a request was made for a room in the hospital. She was also given 100 mg. of Anturitin-S. On the following day the menstrual flow had been reduced by 50%. This treatment was continued for five days when the flow had practically ceased. That night, however, she became acutely ill, vomited, and the menses immediately became more profuse than ever. A fresh crop of purpuric spots developed in the mouth and over the body. She was admitted to the hospital the following day.

Chart No. 1 shows the laboratory findings at this time and up to the day of operation. On the first hospital day she was given 500 cc of citrated blood. That night she developed a severe buzzing sound in the head which required opiates for relief. The following morning she complained of blurring vision and both pupils were dilated and fixed. A neurological examination was negative. Apparently she had had a hemorrhage some place back of the optic chiasma. She was given small daily bood transfusions of 250 cc citrated blood. At the end of a week there was no improvement in the patient's condition. The blood count was practically unchanged, the platelet count lower, the menstrual flow more profuse, the buzzing the head required frequent sedatives, while nausea and vomiting persisted. At this time the family gave their consent for splenectomy to be performed.

A review of the literature reveals some interesting facts:

1. Thrombocytopenic purpura is the No. 1 indication for splenectomy.

2. Not many splenectomies for thrombocytopenia have been reported. Most reports consist of one case and then the essayist procedes to review the other cases that have been reported.

3. One surgeon discouraged the use of preoperative transfusions, stating that it was impossible to improve the blood condition. This observation was confirmed in the case reported here.

4. It was the general impression that as soon as the spleen was removed, the platelet count would rise rapidly and might reach a count of 1,500,000. This did not occur in our case. In fact, the platelet count was very reluctant to rise and changed but little until the patient received a transfusion one week after operaton.

Purpuras are classified as primary and secondary. Secondary purpuras occur in the more severe forms of malignant endocarditis, typhoid fever, typhus fever septicemia, pyemia, whooping cough, measles, cerebrospinal meningitis, and in the more virulent of the tropical diseases. It is also seen in connection with such chronic diseases as scurvy, syphilis, leukemia, pernicious anemia, Hodgkin's disease, various cardiac and nephritic lesions, and in fact with any cachectic or debilitating ailment.

The primary purpuras may be the simple type found chiefly in young children, without any disease or changes in the blood. Then there is the arthritic type of purpuras which are accompanied by joint changes and are found mainly in young adults. Examples are Schonleins Disease and Henoch's Purpura.

Primary idiopathic thrombocytopenia purpura is defined as a morbid state characterized in its graver forms by a deficiency of blood platelets, prolonged bleeding time, deficient contractility of the blood clot without delayed coagulation time, submucous and subcutaneous hemorrhages. It is more prone to occur in young females. There are no reliable figures relating to the mortality in purpura homorrhagica, but it is believed that most cases recover spontaneously. In rare cases known the disease becomes fulminating, with extensive hemorrhage from the mucous and serous surfaces, and death rapidly ensues.

LABORATORY REPORTS PRIOR TO OPERATION

10-19-46: Hg. 4 gms. 28%; coag. time 9 min.; bleeding time 10 min.; RBC 2,160,000; leuk. 8,750. Icteric index: 2 units. Platelet count: 70,000 cu min. Sedimentation rate: 150 mm/60 minutes.
10-20-46: Prothormbin time: 70%
10-21-46: Hg. 27 gm. 29%; RBC 2,160,000; Kahn & Kline negative.
10-23-46: Hg. 5.5 gm. 35%; RBC 2,110,000; leuk. 9,300. Platelet count: 30,000/cu.mm.
10-22-46: Brucellergen test: Immediate reaction,—none; 24 hr. neg.; 48 hr. neg.
10-23-46: Clot retraction: no retraction after 28 hrs. Rh positive.
10-26-46: Platelet count: 63,000 per cu.mm.
Operation performed on 10-27-46.

BLOOD REPORTS FOLLOWING OPERATION

10-28-46: Hg. 6.75 gm. 44%; RBC 2,220,000; leuk. 18,250 platelet count: 52,000/cu.mm.
10-29-46: Hg. 5.7 gm. 38%; RBC 1,910,000. Platelet count: 46,000
10-30-46: Hg. 6.25 gms. 40%; coag. time 4½ min:; bleeding time 3 min.: RBC 2,030,000. Platelet count: 128,000.
10-31-46: Platelet count: 100,000/cu.mm. Bleeding time: 3 min. Clotting time 4½ min. Clot retraction time: no retraction within 30 hours.
11-1-46 Hg. 9 gm. 58%; RBC 3,090,000; leuk. 14,500. Prothrombin time: Control 13 sec.: Patient 15 sec.: Result.—87% of normal. Platelet count: 82,500.
11-2-46 Platelet count: 95,000/cu.mm.
11-3-46: Hg. 10.5 gm. 68%; RBC 3,190,000. Platelet count: 90,000 per cu.mm.
11-4-46: Thrombocyte count: 102,000/cu.mm.
11-5-46: Platelet count: 137,000/cu.mm.
11-6-46: Thrombocyte count: 153,000/cu.mm.
11-7-46: Thrombocyte count: 110,000/cu.mm.
11-8-46: Hg. 6.5 gm. 42%; RBC 2,460,000. Thrombocyte count: 140,000/cu.mm.
11-9-46: Thrombocyte count: 150,000/cu.mm.
11-11-46: Hg. 6.5 gm. 42%; RBC 2,290,000 Thrombocyte count: 92,000/cu.mm.
11-12-46: Thrombocyte count: 112,000/cu.mm.
11-13-46: Thrombocyte count: 119,000/cu.mm.
11-19-46: Hg. 8.5 gm. 55%; RBC 2,730,000. Thrombocyte count: 92,000/cu.mm.
11-22-46: Platelet count 117,000/cu.mm.
11-27-46: Platelet count: 93,000/cu.mm.
12-1-46: Hg. 10.5 gm. 68%; RBC 2,910,000. Platelet count: 112,000/cu.mm.
1-10-47: Platelet count: 180,000.

Thomas W. Woodman, M. D.—

This case was extremely interesting to us because of the rather unexpected clinical course following splenectomy. We were quite certain that this patient had a primary type of thrombocytopenia purpura and that following the splenectomy the platelet count would increase so rapidly that it would probably be necessary to give some anti-coagulants in order to prevent clotting. As a matter of fact it is common to have this platelet increase begin almost immediately on ligation of the splenic artery. We watched anxiously for the increase in platelets but, as will be noted on the record of blood counts following surgery, this increase did not promptly occur, and as a matter of fact the platelet count was not particularly good at the time of discarge from the hospital. The convalescence following splenectomy was uneventful as far as the operation was concerned. However bleeding slowed, stopped, and then recurred to some extent. Hemorrhage was not however particularly severe at any time following the operation and it had been terrific immediately prior to surgery.

We gave two pints of blood just prior to surgery and another pint immediately afterward. Bleeding was not severe during the operative procedure and the operation itself did not present any particularly unusual technical difficulties. Recovery was somewhat slow but on the whole progressive and the patient returned to her home in apparently good condition except for the fact that the platelet count was low. She was in the office about a week ago and at that time the platelet count was one hundred eighty thousand. She felt perfectly well except for visual disturbance. This, she stated, was slowly improving. She had a normal menstrual period recently.

Considerable has been written on the importance of sternal puncture in the differentiation of the primary and secondary thrombocytopenia. Sternal puncture was not done in this particular case for it was believed, after consultation with pathologists, that the differentiation would not be absolute and that the condition of the patient was such that if there was the slightest doubt she should be given the benefit of splenectomy. I do not feel that she would have recovered without splenectomy but I'm still not completely convinced as to whether this case was one of primary or secondary thrombocytopenia.

There are certain blood dyscrasias that respond miraculously to surgery and others for which surgery gives palliative relief, and there are still others for which surgery offers nothing. The surgery in these dyscrasias is limited mainly

to splenectomy. In Familial Hemolytic Anemia, Idiopathic or Primary Thrombocytopenic purpura, and Banti's disease, splenectomy offers excellent results. Palliative relief is obtained by splenectomy in Gaucher's disease and Von Jaksch's anemia.

We are primarily interested tonight in the Idiopathic thrombocytopenic purpura or Werlhof's disease. In this condition there is marked and uncontrollable reduction in the number of platelets, spontaneous hemorrhages of the mucous membrances and purpura. Giant platelets are commonly observed in the infection. The disease most commonly attacks young delicate girls, may follow an acute infection or tuberculosis. Spontaneous capillary bleeding from and into the mucous membranes, especially the nose and mouth are the most frequent sites. Bleeding from the uterus is common and hemorrhages from the retina and kidney are rather common. The subcutaneous hemorrhage as a rule is not profuse but usually consists of a few petechiae not associated with any exudative lesions. The blood pressure cuff test of diminished capillary resistance is positive and is roughly proportional to the degree of platelet reduction. Characteristically the bleeding time is prolonged, but the rate of clotting in vitro is normal. In acute cases there is an abrupt onset without warning or there are a few days of general debility. In favorable cases the purpura clears spontaneously within three weeks. Intermittant cases recur at intervals of days or weeks, and most cases die within a few months. Chronic cases do not develop from acute cases. The secondary form of purpura with thrombocytopenia is due to some recognized disease as a rule acting by producing extensive changes in the bone marrow and thereby disturbing production of the platelets from the megakaryocytes. Examples are tumors such as marrow carcinomatosis or other tumors and blood diseases such as aplastic anemia, pernicious anemia, leukemia and granulopenia. Severe infections such as sepsis typhoid and acute hemorrhagic smallpox may result in secondary thrombocytopenia. Intoxications from benzene, aniline, ANA, arsenic etc., together with radio-active substances, may cause secondary thrombocytopenia. Considerable has been written regarding allergy as a causative factor.

We believed this case to be one of primary thrombocytopenia because of the absence of factors producing the secondary type; together with its severity and the mode of onset. I have been unable to find in the literature any primary thrombocytopenic cases in which the platelet count did not return to normal within a few days following splenectomy. We are keeping our fingers crossed in this particular case but she certainly does seem alright now. I feel perfectly satisfied with having done a splenectomy in this patient and feel that if one is not completely certain that the disease is of a secondary type the patient should be given the benefit of the doubt and splenectomy done.

D. T. T. Frost:

Tissue: Spleen.

Gross Specimen is a spleen weighing 250 grams and measuring 13.0 x 8.0 x 6.5 cms. The capsule is moderately thick, the pulp is reddish brown in color and the malphighian nodules are fairly large and distinct.

Microscopic The capsule and trabecullae of the spleen are not unusual. The pulp spaces show considerable hyperplasia of the cells, but the lumen of the pulp spaces is not filled with cells as is usually found in a marked hyperplasia of the spleen. There are scattered polymorphonuclear leukocytes in the spaces. In addition the malpighian bodies are markedly hyperplastic with large germinal centers, and more thickness of lymphocytes surrounding the secondary nodules. The blood vessels are not unusual.

Comment: While this spleen is consistant with thrombocytopenic purpura, there are no characteristic findings in the spleen of this disease. However, there is one disturbing element, and that is the presence of the polymorphonuclear leukocytes in the pulp spaces suggestive of inflammatory reation. However, this is not acute in character, and may be due to the increase irritation from operative procedures. No abscesses or other suggestion of an acute splenitis is present.

Pathological Diagnosis: Hyperplasia of the spleen particularly of the lymph follicles (consistant with thrombocytopenic purpura).

DISCUSSION

Grossly and microscopically this spleen is typical. The hyperplasia of the germinal centers of the lymph follicles and the appearance of a megakaryocyte in one of the sinusoids substantiate the clinical diagnosis. The necrosis of the germinal centers sometimes found is not present in this spleen.

In essential thrombocytopenic purpura, the spleen is notable for its lack of specific change and it is impossible to differentiate this spleen by objective criteria.

The history and laboratory findings and the changes in the spleen of this case fit well into the picture of essential thrombocytopenic purpura.

THE PEPTIC ULCER PROBLEM

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THE etiology and treatment of few diseases have caused more comment than those of gastric and duodenal ulcer, and of all of the various types of treatment yet mentioned for peptic ulcer none to my knowledge has met with complete unanimity of the medical profession.

I think that it can be safely stated that gastric ulcer is a surgical condition unless it quickly responds to medical treatment. In this mention of response to medical treatment I have reference to the group of cases of gastric ulcer in which roentgenographically the lesion and its symptoms disappear after a few weeks of medical management during which time the patient is hospitalized. All other cases are surgical. There is no doubt, any more, that some persistent gastric ulcers undergo malignant change.

The big problem, as I see it, is how best to manage duodenal ulcer. This lesion does not become malignant but because of the frequency with which it occurs and the debilitation it produces, constant search is being made for a satisfactory method of treatment which can be employed in the management of the intractable type of ulcer. My personal opinion is that the majority of duodenal ulcers can be well controlled or healed by good medical management.

About 13 per cent of the cases in which duodenal ulcer is examined at the Mayo Clinic are considered surgical. These ulcers are classified as intractable: they have not responded to medical management, produce severe pain and cause obstruction or periodic hemorrhages.

I am of the opinion that some surgeons consider the majority of patients with duodenal ulcer candidates for surgical treatment. From my observation, I am prompted to ask: Can more of these patients not be treated medically? Sippey, you will recall, considered most duodenal ulcers as medical problems. It was he who laid down the dictum that to heal peptic ulcer the gastric acids must be constantly neutralized or kept at a low level. Some of the present-day methods of medical management of ulcer, in my opinion, have taken on a C.I.O. hue in that the medical treatment is carried out religiously during the daytime while during the night the patient is told "to get his rest." Such management fails utterly to take into consideration that the duodenal ulcer patient's stomach is unable to tell day from night. Therefore, as we all know, the outstanding feature of the condition is night pain which awakens the patient and he then obtains relief by the ingestion of food. Adequate medical therapy therefore requires "around the clock" treatment.

When is a duodenal ulcer a surgical problem? The present consensus would seem to be that operation for ulcer of the duodenum is justified and indicated when characteristic ulcer pain, pyloric obstruction or hemorrhage persists or recurs in spite of adequate medical management, assuming surgical intervention is necessary. What type of procedure should be carried out? This question has received, as you well know, many answers. Some surgeons think gastroenterostomy is sufficient. Others think gastric resection, removing two thirds of the stomach with or without removal of the duodenal ulcer, is the procedure of choice. In 1943 Dragstedt after exhaustive animal experimentation, elected to try interrupting the vague nerves immediately above the diaphragm by a tranthoracic approach or by a subdiaphragmatc or abdominal approach for intractable duodenal ulcer.

The vagus nerve contains motor, secretory, vasodilator and sensory fibers. Its main functions from a physiologic standpoint seem to be motor and secretory. If one considers the neurogenic factor an important one in the etiology of duodenal ulcer, it would seem that with interruption of both vagi in the region of the lower end of the esophagus the hydrochloric acid units in the gastric juice would be lowered or abolished. Dividing the vagus nerves below the heart with

Read before the Lois Grunow Memorial Clinic, Phoenix, Arizona, February 20, 1947.

the hope that the procedure might bring about an abatement of acid secretion from gastric mucosal cells has been carried out by many investigators with variable results. Brodie in 1814 attempted to denervate the stomach by sectioning the vagus nerves. Laterjet in 1922 divided the "gastric nerves" in dogs. He found the emptying time of the stomach delayed, gastric acidity reduced and the secretion of mucus increased. He divided the vagus nerves in six patients suffering from gastric ulcer and performed gastrojejunostomy at the same time. Five, he stated, were relieved. One patient later died because of gastric carcinoma. Other well-known experiments on the vagus nerves and their possible influence on gastric secretion have been made by Ivy, Dragstedt, Walpole, Hartzell, Carlson, Hollander, Mann and others.

The effect on the secretory mechanism of gastric mucosa as to amount and acid content of secretion after vagus section has not produced identical results in the experimental animal or in man by the various investigators. It is sufficient to say, however, that interruption of the vagus nerves at the lower end of the esophagus lends credence to the belief that such procedure does bring about a decrease in the acid secreted by the gastric mucosa.

It has been long observed that duodenal ulcer is practically nonexistent in people with achlorhydria. This observation, coupled with the relief obtained in patients suffering from duodenal ulcer on the oral administration of antacid substances, logically brings to mind a statement made by Dragstedt that perhaps peptic ulcer should be called "acid ulcer."

Sixty years ago, Joseph Warren asked: Why does not the gastric juice digest its own wall since other albuminous substances are readily acted on? John Hunter said that the stomach did not digest itself because of the "living principle" which exists in live tissue. He further postulated that if it were possible to place the hand of a living man into the stomach of a lion, the gastric juices would not cause the hand to be dissolved but if the hand were that of a dead man, surely it would be dissolved. He further emphasized again that, because of the "living principle," gastric secretions would not destroy living tissue as would an acid placed on one's skin surface. He continued by citing that living animals, such as worms, flies and maggots were not digested when taken into the stomachs of other animals. Pavlov and Heidenhain constructed accessory stomach pouches in order to study the effect of pure fundus secretions.

Dragstedt and Ellis isolated the entire stomach in dogs by anastomosing the duodenum to the lower part of thre esophagus. In this study they carefully left intact both vagi as well as the blood vessels supplying the stomach. Gastric juice in such an isolated stomach is secreted by means of impulses carried by the vagus nerves and brought about by the hormone gastrin which normally is liberated by the stomach and upper part of the intestine after ingestion of food. The fundus of such an isolated stomach secreted an aqueous solution of hydrochloric acid and enzyme pepsin. It is of interest that Dragstedt and Ellis in these experiments found a constant acid concentration of 135 units. If living tissues such as the spleen or kidneys, with blood supply intact, were implanted in accessory stomachs, ulceration of the implanted organs soon occurred and death of the animal took place witwhin a few days owing to hemorrhage.

If such an experiment were carried out implanting the kidneys, pancreas or a segment of the ileum into the normal stomach of the experimental animal, the implanted organ with its blood supply intact became covered by gastric mucosa but did not show evidence of ulceration. These experiments obviously emphasize that in the nonisolated stomach the buffer action of the pancreatic, duodenal and biliary secretion regurgitated into the stomach prevented digestion and ulceration of implanted living tissues. In these experiments on the intact stomach ulceration of its mucosa produced by trauma, corrosives or other factors invariably healed without treatment. From these and numerous other studies one might assume that peptic ulcer occurs in man when an excessive secretion of gastric juices occurs. If the foregoing is true, what then is the treatment of peptic ulcer, particularly duodenal ulcer of the intractable type? If excessive gastric juice is of neurogenic origin, what effect does vagotomy have on peptic ulcer in man?

In answer to these questions one must first state that vagotomy cannot fully be appraised until more time has elapsed. Those of us who have resected a segment of both vagus nerves have made certain similar observations. First immediately following operation there is an absence of ulcer pain, a marked lowering of the hydrochloric content of the gastric juice and a

decrease in the amount of gastric secretion. On the basis of my personal experience of some twenty-five to thirty cases of resection of a portion of the vagus nerves in ulcer patients, I am of the present opinion that the procedure should be carried out by an abdominal approach which permits exploration of the stomach and duodenum. If obstruction is present, as is frequently the case, in addition to segmental vagus resection at the lower end of the esophagus, gastro-enterostomy or gastroduodenostomy should be done. These combined procedures may mask for at least a time the neurogenic factor because it is known that gastro-enterostomy or gastroduodenostomy will in a certain percentage of cases give at least good temporary results. Gastro-enterostomy in elderly patients with obstructive surgical duodenal ulcer in which the concentration of acid is not high, may, as we know, give permanent relief. In three cases of gastrojejunal ulcer and one gastrojejunocolic fistula, only vagotomy was performed. There was immediate relief of all symptoms. In the case of gastrojejunocolic fistula the opening communicating with the colon, stomach and jejunum was closed, the gastrojejunal stoma left intact and a portion of each vagus removed. The patient died suddenly four or five months later from coronary occlusion. During the interim between the operation and his death, he had according to his referring physician, no ulcer symptoms. At the time when this paper was written, the other two patients were experiencing complete relief from their previous ulcer symptoms.

All but two of the patients having duodenal ulcer on whom I have personally operated have had either pyloric obstruction, recurring hemorrhages or both. In addition to vagotomy I have carried out gastrojejunostomy or gastroduodenostomy. The two cases in which vagotomy alone was performed were among my early cases; in both there was moderate duodenal obstruction and in retrospect some type of new gastric outlet should have been made at the time vagotomy was performed. One of the patients required gastrojejunostomy, which I established four months later. When this paper was written, fifteen months later, this patient had gained 40 pounds (18.1 kg.), ate as he chose and was free of peptic ulcer symptoms. The other patient had intermittent gastric retention and vomiting but he has gradually improved and has no ulcer pain. It is interesting to emphasize here that, even though these two patients had even more gastric obstruction following the initial operation (vagotomy) than they had primarily, they had no return of ulcer pain and their gastric acid content remained low and showed little or no presence of free hydrochloric acid.

Vagotomy produces at least for a time an atonic stomach as emphasized by Dragstedt, who found it necessary to carry out gastro-enterostomy in seven cases secondary to division of the vagus nerve in his first fifty or sixty cases.

At the Mayo Clinic during the eighteen months prior to January 1, 1947, vagotomy has been performed in eighty-three cases. The majority of these procedures were performed by a subdiaphragmatic or abdominal approach and in only fourteen was vagus nerve interruption alone done. In all others gastro-enterostomy had already been performed as previously mentioned, or gastro-enterostomy or gastroduodenostomy was done at the time of vagus section.

Various studies have been carried out on most of the cases in which we have performed bilateral vagotomy. In the majority of cases the amount of night secretion was diminished. Also, there was produced almost invariably a decrease in the hydrochloric acid content of the gastric secretions or an achlorhydria. The postoperative administration of histamine to the patients after the method of Code produced a marked rise in the gastric contents of free hydrochloric acid, which one would expect, since acid-secreting cells of the stomach were still present.

Hollander suggested the administration of insulin to patients following vagus section. His studies are based on the principle that if 10 to 30 units of insulin be given such patients tomporary hypoglycemia is produced and if during this hypoglycemic state the gastric contents show the presence of hydrocholoric acid, it signifies that the caudal nucleus, from which the vagus nerves arise, are still capable of sending impulses through the vagus nerves, thus causing the acid-secreting cells to function and therefore all the vagus nerve branches have not been severed.

To summarize briefly: What is the present status of vagotomy in the treatment of gastric or duodenal ulcer?

First, it seems fair to state that the neurogenic factor may play an important role in the causation of peptic ulcer since the condition occurs most often in highly nervous, excitable persons. Vagotomy does immediately relieve the ulcer

muscles. The esophagus is freed from the surtype of pain in almost every case; from the vast amount of experimental evidence such relief would seem likely to be due to the lowering or abolition of hydrochloric acid content of the gastric secretion. One of my colleagues, Dr. W. W. Walters, is of the opinion that at the present the procedure is in the state of indecision.

To procedure, vagotomy, along with an additional stomach outlet seems to me worthy of further trial in preference to gastric resection for duodenal ulcer. The risk of such operative procedures is not high. Three deaths have occurred in the hospital postoperatively, one from coronary occulsion and from subhepatic abscess due to perforation of a duodenal ulcer which occurred prior to vagotomy and gastrro-enterostomy. The third death, though no necropsy was carried out as was done in the two preceding cases, occurred on the fifth postoperative day during a convulsion thought to be due to a cerebral vascular accident.

Finally, my opinion may be summed up as follows:

1. Section of the vagus nerves is best carried out by means of an abdominal approach. Personally, I employ an oblique upper abdominal incision which extends from the right costal margin to within 3 or 4 inches (8 or 10cm.) of the left superior iliac crest, dividing both rectus rounding structures, a rubber tube is then placed around the cardia and the esophagus is drawn downward a distance of 2 or 3 inches (5 or 8 cm.). Both vagus nerves are easily palpated and resemble taut violin strings. On visualization a section of both nerves is removed 1 to 2 cm. in length.

2. As a rule, especially if duodenal obstruction is present, some type of additional stomach outlet should be made, as previously mentioned.

3. In cases of gastric ulcer it seems wise to remove the leerated area. If the lesion is malignant, radical gastrectomy is in order.

4. Perhaps time will prove that poor results such as recurrence of peptic ulcer following the foregoing operation is due to the presence of small branches of the vagus nerve which have not been severed or have been only partially removed.

5. My experience thus far in this method of management of ulcer is similar to that of Dragstedt who after carrying it out on 170 patients with various types of peptic ulcer is of the impression that it seems physiologically sound but that more time and study on the patients thus treated is required before a definite and final statement regarding it can be made.

THROMBOSIS AND THE ANTICOAGULANTS

OVID O. MEYER, M. D.**
Madison, Wisconsin

IN 1936 Charles and Scott[1], working in Best's laboratory in Toronto, obtained a cry stalline form of heparina mucoitin polysulfuric acid pure enough to be administered to human beings. Previous to this, the impurities in heparin that had been isolated prevented its employment as an in vivo anticoagulant. Much attention to the role of the anticoagulants in clinical medicine followed the investigations of Charles and Scott[1], and Murray and Best[2].

The investigation of thrombosis and its prevention and treatment was further stimulated in 1939 by the isolation of an entirely different anticoagulant derived from spoiled sweet clover and later synthesized by Linq[3] and his associates. This substance, 3,3'-methylenebis (4-hydroxycoumarin), which is distributed under the trade name of Dicumarol, operates in a manner quite different from heparin in lessening the incidence of thrombosis.

The theory of coagulation most widely accepted at the present time may be expressed in the following formula:

Prothrombin + Ionized Calcium + Thromboplastin = Thrombin

Thrombin + Fibrinogen = Fibrin

The prothrombin is derived principally, if not solely, from the liver. The thromboplastin is present in many body tissues, the platelets being an important source. The fibrinogen is synthesized in the liver. Depletion or reduction of any

*Read as a lecture in Medical Sciences, Lois Grunow Memorial Clinic, Phoenix, Arizona, February 22, 1947.

**From the Department of Medicine, University of Wisconsin Medical School.

of these three elements will cause delay in coagulation of the blood and any interference with the chemical process will also reduce its coagulability. Intravascular clotting is prevented, apparently, by the small amount of thromboplastin present and by an antithrombin which neutralizes any thrombin formed. This antithrombin is apparently a serum albumin.

A thrombus may be white, made up essentially of platelets, or red, composed largely of fibrin and red blood cells entangled in it, although there is always a basis of platelets. Mixed thrombi, partly pale and partly red, are the most common. A thrombus and a coagulum are closely related and for practical purposes need not be differentiated, particularly since mixed thrombi are the most frequent and since it is accepted that thrombus formation is inhibited by hypocoagulability of the blood.

There are several possible etiologic factors in thrombus formation. The starting point of a thrombus is a small mass of platelets which usually has been preceded by damage to the vessel walls, stasis of the blood, or a modification of the coagulability of the blood. The common factors that may be involved are: (1) trauma; (2) infection; (3) chemicals; (4) stasis, local or cardiac; (5) thrombophilia; (6) drugs, such as digitalis, aminophyllin, snake venoms, etc.

A blow to a part of the body, as the leg, for example, may injure a vein and result in endothelial damage that might cause a thrombus to develop at the site. Infection (phlebitis) is commonly associated with thrombus formation because of damage to the vessel, a form of trauma in a sense. Similarly, chemicals injected into a vein, especially if they are not rapidly carried off by the flow of blood may result in damage to the venous wall with resultant thrombosis at the site. One immediately thinks of many chemical irritants capable of this, such as a hypertonic glucose, bichloride of mercury, dyes injected for visualization of the gall bladder, penicillin, and many others.

Stasis of the blood is also a frequent cause of thrombosis. This may be local where there is obstruction of the venous return from an extremity or other part. or, much more frequently, it may be general, as in cardiac decompensation. Several years ago Dr. Mead Burke[4] observed from autopsy findings that the cases of venous thrombosis from the medical service exceeded those from the surgical service, and that the incidence was extremely high in patients that had had cardiac decompensation. It was especially frequent during the first two weeks of hospitalization, that is, during a period of immobilization with bed rest.

Thrombophilia, a great increase in platelets after splenectomy, for instance, is very conducive to the development of thrombosis, and it seems to me that the administration of an anticoagulant after this operation is clearly indicated. Finally, the work of Macht[5], of De Takats, Trump and Gilbert[6] and of others suggests that the administration of digitalis produces hypercoagulability of the blood and thus leads to thrombus formation and embolism. More recently, Link[7] and his associates have demonstrated that the methyl xanthines, caffeine, theobromine, and theophyllin may produce hyperprothrombinemia in animals, and, they infer, a greater likelihood of thrombosis in man, although clinical evidence for this inference is yet wanting. In 1945, Quick[8] reported before the Central Society for Clinical Research that he was unable to establish that the methyl xanthines had a hyperprothrombinemic action, and Rieben[9] of Switzerland has also reported failure to confirm Link's observation. It has long been known that several snake venoms may lead to thrombus formation in some instances by acting on prothrombin to form thrombin, and in others, as with the moccasin venom, by acting directly on fibrinogen to form fibrin. Cobra venom, on the hand, is an anticoagulant.

Many anticoagulants are well known, some of which act only in a test tube, others in vivo as well as in vitro; Dicumarol is useful only as an in vivo anticoagulant.

Some of the commoner anticoagulants are: (1) oxalates, which prevent clotting in a test tube by precipitating calcium; (2) fluorides, which form a weakly dissociated calcium salt; (3) sodium citrate, which forms a double salt, calcium sodium citrate; (4) azo dyes, such as trypan blue, and Chicago blue 6b. These are all used to prevent coagulation in a test tube. The anticoagulants most commonly used for preventing clotting in vivo are (5) hirudin, the anticoagulant of leeches, which acts as a typical intithrombin like heparin in that it inactivates thrombin; (6) snake venom, notably cobra venom, a powerful anticoagulant which probably acts on the thromboplastin to produce its effect, in association with erythrocyte hemolysis; (7)

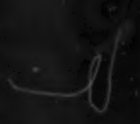
heparin, a mucoitin polysulfuric acid and an extremely potent anticoagulant derived from beef liver and other tissues, which acts as an antithrombin preventing conversion of prothrombin to thrombin: According to Quick[10], its acid properties allow it to bind proteins or a protein of blood, which is the explanation of its antithrombic activity. The complex so formed has the power to bind thrombin, which is then prevented from performing its essential role in the coagulation of blood. This reaction of protein binding is reversed by a strongly basic protein like protamine, which is an effective antidote for an excess of heparin. Heparin also has the property of diminishing the agglutinability of platelets, a very important function in the prevention of thrombosis, Wright[11], Best, Cowan and MacLean[12], and Dale and Jaques[13].

Heparin is quantitated in units. Although a unit is not exactly defined, it is usually considered to be the amount of the anticoagulant which inhibits the clotting of 1 cc. of blood in the cold for 24 hours. Best[14] has suggested that a unit might profitably be defined as the activity contained in 1/100 mg. of the purest material. Not all products on the market are of the same concentration, but some are so prepared that 1 cc. is the equivalent of 10 mgs. or 1000 toronto units of heparin; thus a 10 cc. vial would contain 10,000 units. For the average case, it is custmary to put 20 cc. in the initial liter of glucose or saline, allow the fluid to flow slowly, and measure the coagulation time at two ot four hour intervals until the proper coagulation time is reached, i.e. 15 to 20 minutes, the two-tube method of Lee and White being used to determine the coagulation time. After this is attained, the flow is adjusted to maintain this level; 10 cc. in each subsequent liter is usually adequate, the fluid perhaps being allowed to flow somewhat more rapidly to maintain the proper coagulation time. The heparin must be given parenterally, preferably intravenously, and, in the opinion of most workers, preferably continuously rather than intermittently. Jorpes[15], however, administers heparin intermittently and feels that this is the proper procedure. Two to four hours after the administration of heparin is stopped, the coagulation time of the blood returns to normal.

Dicumarol, 3,3′-methylenebis (4-hydroxycoumarin) is the synthesized material which is biologically identical with the material isolated from spoiled sweet clover responsible for hemorrhagic disease in cattle. It reduces the prothrombin in the blood[16] and, as we have found recently, also lessens the agglutinability of platelets[17] and produces hypocoagulability of the blood in man and in many animals. It does not function as an anticoagulant in vitro. Its great advantage over heparin is that it is inexpensive and is active when given orally, irregularly active when given rectally. It can be given intravenously in a strongly alkaline solution, which is not as stable as might be desired. Dicumarol produces no demonstrable effect upon the prothrombin time for 24 to 48 hours after administration, and the effect is protracted, reaching a maximum in 3 to 5 days and then subsiding for 2 to 4 days until return to normal. This protracted effect, it is obvious, may be either advantageous or disadvantageous, according to circumstances. The usual initial dose is 5 mgs./Kg. Thereafter we usually give 1.5 mgs./Kg. per day, the dosage being determined by the prothrombin time; when it is too great, the drug is omitted one or more days until the prothrombin time starts to decrease, when the 1.5 mgs./Kg. dosage, as needed, is[6] resumed. It is obvious that it requires some experience and skill to judge when the drug should be continued and when ommitted. We aim to maintain the prothrombin time at approximately 25 to 50 per cent of normal — that is, in a range of 16 to 21 seconds by our method of determining prothrombin time, where the control time is about 11 seconds for a prothrombin of 100 per cent. With the suggested dosage 80 per cent of patients will obtain desirable prothrombin levels. Some require larger doses, and an occasional patient is very sensitive to the drug. Consequently, individualization of therapy and daily determinations of prothrombin time, at least for the first week, and frequently thereafter, are absolutely essential if accidents are to be avoided.

Dicumarol should not be administered to patients with hemorrhagic disease, significant liver disease, or renal disease with gross impairment of function. Caution is indicated if fever or a dietary deficiency exists.

The antidotes, if excessive hypoprothrombinemia or bleeding occur, are transfusion of fresh blood, perhaps daily for several days. 400 to 500 cc. and/or the intravenous administration of large doses of synthetic vitamin K, 40 to 64 mgs. Ordinary clinical doses of Vitamin K are totally ineffectual.



Fifty-sixth Annual Meeting

ARIZONA STATE MEDICAL ASSOCIATION

TUCSON, ARIZONA

May 7-10
1947

Headquarters:
HOTEL PIONEER

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Official Call

THE ARIZONA STATE MEDICAL ASSOCIATION and the AUXILIARY TO THE ASSOCIATION convene in annual session at Tucson, May 7-10, 1947. The usual scientific and business sessions will be held, the Hotel Pioneer serving as headquarters for the sessions.

It is a pleasure to announce this session.

George O. Bassett

President
Arizona State Medical Association

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MEETING PLACES - HOTEL PIONEER

Medical Association

Registration ..... Mezzanine
Council Sessions ..... Varsity Room
House of Delegates ..... Varsity Room
Scientific Sessions ..... Ball Room
President's Dinner Dance ..... Ball Room

Woman's Auxiliary

Registration ..... Lobby
Sessions ..... Mezzanine
Luncheons and Dinners ..... To be announced

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SCHEDULE OF BUSINESS AND ENTERTAINMENT

Medical Association

Registration (each day) ..... May 7 - 10
Council Sessions ..... May 7 at 1:00 P. M.
Called Sessions as set

House of Delegates ..... May 7 at 4:00 P. M.
Called Sessions as set

President's Dinner and Dance..May 8 at 7:30 P. M.
Pioneer Hotel Ball Room
(Open to members and ladies)

Woman's Auxiliary

Business Sessions, time and place of Luncheons and Dinners to be announced.

ARIZONA STATE MEDICAL ASSOCIATION OFFICERS AND COUNCIL

George O. Bassett ..... President
Prescott
Preston T. Brown ..... President-Elect
15 E. Monroe, Phoenix
Robert E. Hastings ..... Vice-President
1811 E. Speedway, Tucson
Frank J. Milloy ..... Secretary
15 E. Monroe, Phoenix
C. E. Yount ..... Secretary
Prescott
Harold W. Kohl ..... Speaker of House
1811 E. Speedway, Tucson
Jesse D. Hamer ..... Delegate to A. M. A.
15 E. Monroe, Phoenix
D. F. Harbridge ..... Medical Defense
15 E. Monroe, Phoenix

COUNCILORS

Robert S. Flinn ..... Central District
Arthur C. Carlson ..... Northern District
Hal W. Rice ..... Southern District

COUNCILORS AT LARGE

O. E. Utzinger ..... Ray
Dan L. Mahoney ..... Tucson
W. Paul Holbrook ..... Tucson

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COMMITTEES
ANNUAL MEETING

Committee on Scientific Assembly
Preston T. Brown, President-elect and Committee Chairman (1947), Phoenix; H. T. Southworth (1949), Prescott; Carl H. Gans (1947), Morenci; Robert E. Hastings (1948), Tucson.

Registration:
Frank J. Milloy, Chairman ..... Phoenix

House of Delegates Committees:
To be announced.

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Guest Speakers

EMIL NOVAK, M. D., F.A.C.S.
Baltimore, Maryland
Associate in Gynecology, Johns Hopkins University Medical School and Hospital.

GEORGE T. PACK, M. D., F.A.C.S.
New York City, N. Y.
Attending Surgeon Memorial Hospital, New York City
Assistant Professor Clinical Surgery, Cornell University, New York City

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Program

Thursday Morning, May 8

GENERAL SESSION

9:30 A. M.
Call to Order
Geo. O. Bassett, M. D., President
Invocation
Welcome ..... V. A. Smelker, M. D.
Response ..... E. A. Born, M. D.
Induction of President
Geo. O. Bassett, M. D., officiating.
Presidential Address
Preston T. Brown, M. D., Phoenix
Memorial Service
Hal W. Rice, M. D., Historian

10:15 A. M. SCIENTIFIC

1. "Incidence of Certain Diseases Among the Navajos"
   Clarence G. Salsbury, M. D., Ganado, Ariz.
   Discussion
2. "Medical Defense—In Review"
   C. E. Yount, M. D., Prescott
   W. W. Watkins, M. D., Phoenix
   D. F. Harbridge, M. D., Phoenix

Thursday Afternoon, May 8

1. "The Use of Potassium Chloride in Treatment of Dehydration and Diarrhea in Infants."
   A. I. Podolsky, M. D., Yuma
2. "Hypertrophic Pyloric Stenosis and Vomiting in Infancy."
   Donald E. Nelson, M. D., Safford
   Discussion Period on Above Two Papers.
3. "Sex, the Great Taboo — a Gynecological Study"
   Arnott K Duncan, M. D., Douglas
4. "Management and Mechanism of Functional Uterine Bleeding."
   Emil Novak, M. D., Baltimore, Maryland
   Question and Answer Period on Above Two Papers.

PRESIDENT'S DINNER-DANCE

Ball Room, Pioneer Hotel.....7:30 P. M.

Friday Morning, May 9

9:30 A. M. SCIENTIFIC

1. "The Treatment of Varicose Veins"
   H. D. Cogswell, M. D., Tucson
2. "Cancer Prevention"
   E. Payne Palmer, M. D., Phoenix
3. "Carcinoma of the Rectum"
   W. R. Manning, M. D., Tucson
4. "The Management of Malignant Melanoma"
   George T. Pack, M. D., New York City
   Question and Answer Period on Above Papers.

Friday Afternoon, May 9

2:00 P. M.

1. "Medical Observation on the Management of Shock."
   Charles A. L. Stephens, M. D., Tucson
   Discussion
2. "Epidemic Hepatitis"
   Joseph Bank, M. D., Phoenix
   Discussion
3. "Management of Patients with Bronchial Asthma."
   F. B. Schutzbank, M. D., Tucson
   Discussion
4. "Recent Advancements in the Treatment of Heart Disease"
   Robert S. Flinn, M. D., Phoenix
   Discussion
5. "Hydatidiform Mole Followed by Chorio-epithelomia."
   Report of a Case.
   Clarence B. Warrenburg, M. D., Phoenix

Friday Evening, May 9

7:30 P. M.

1. "Radical Surgical Treatment of Cancer of the Stomach"
   George T. Pack, M. D., New York City
2. "Indications, Limitations, and Hazards of Endocrine Therapy in Gynecology."
   Emil Novak, M. D., Baltimore, Maryland
   Question and Answer Period on Above Two Papers

WOMAN'S AUXILIARY

ANNUAL MEETING

President—Mrs. Hervey Faris.....Tucson
Pres.-Elect—Mrs. Harry Southworth.....Prescott
First Vice-Pres.—Mrs. Thomas H. Bate....Phoenix
Second Vice-Pres.—Mrs. Joy A. Omer.....Tucson
Recording Sec'y—Mrs. Louis G. Jekel.....Phoenix
Treasurer—Mrs. Karl S. Harris.....Phoenix
Cor. Sec'y—Mrs. Charles E. Starns.....Tucson
Directors—Mrs. Edward M. Hayden, Tucson; Mrs. James H. Allen, Prescott; Mrs. Paul Henry Case, Phoenix.

CONVENTION COMMITTEES

Pima County Auxiliary, Hostess

General Chairman—Mrs. Harold W. Kohl
Vice-Chairman—Mrs. Charles E. Starns

Registration and Credentials

Mrs. Roy Hewitt, Chairman
Mrs. Earl Brown
Mrs. Clyde Flood
Mrs. John Mikell
Mrs. Raymond F. Oyler

Courtesy

Mrs. Charles E. Starns, Chairman

Transportation

Mrs. Edward M. Hayden, Chairman
Mrs. B. B. Edwards
Mrs. Raymond F. Oyler
Mrs. Royal Rudolph
Mrs. Redford Wilson

Telephone and Reservations

Mrs. Arthur J. Present, Chairman
Mrs. Thomas H. Cates
Mrs. Charles A. L. Stephens, Jr.
Mrs. Charles Newcomb

Luncheon Hostesses

Mrs. Harold W. Kohl, Chairman
Mrs. Royal Rudolph
Mrs. W. G. Shultz
Mrs. J. E. Gains

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AUXILIARY PROGRAM

THURSDAY, MAY EIGHTH

10:00 A. M. to 4:00 P. M.

Registration.....Lobby of Hotel Pioneer

12:00 Noon

Luncheon for Board Members.....
El Rio Golf and Country Club

2:00 P. M.

Board Meeting.....Mezzanine, Hotel Pioneer

3:00 P. M.

Mrs. Hervey Faris, President, Presiding

General Session.....Hotel Pioneer
Invocation.....Rev. Leland H. Koewing
Address of Welcome.....Mrs. Charles E. Starns
Tucson
Response.....Mrs. Clarence Gunter, Globe
Speaker—Dr. Emil Novak, Baltimore
Subject .....“Cancer in Women”
Business Sessions:
Roll Call
Reading of Minutes
Report of Meeting of Board of Directors
Reports of State Officers and Committee Chairmen—
President.....Mrs. Hervey Faris, Tucson
First Vice-Pres...Mrs. Thos. H. Bate, Phoenix
Second Vice-Pres.....Mrs. Joy A. Omer, Tucson
Cor. Sec'y.....Mrs. Charles E. Starns, Tucson

Treasurer.....Mrs. Karl S. Harris, Phoenix
Legislative.....Mrs. W. F. Schoffman, Phoenix
Nat'l Board....Mrs. Jesse D. Hamer, Phoenix
Publicity..Mrs. Thos. A. Hartgraves, Phoenix
Bulletin.....Mrs. J. L. Donahue, Tucson
Hygeia.....Mrs. Ludwig Lindberg, Tucson
Health.....Mrs. C. R. Swackhamer, Superior
Post-War Plan'g..Mrs. Alvin Kirmse, Prescott
Historian.....Mrs. George B. Irvine, Tempe

Reports of County Auxiliary Presidents—
Gila.....Mrs. Clarence Gunter, Globe
Maricopa.....Mrs. George S. Enfield, Phoenix
Pima.....Mrs. Joy A. Omer, Tucson
Yavapai.....Mrs. Joseph P. McNally, Prescott

Report of Registration and Credentials Committee—

Report of Nominating Committee—
Mrs. Paul Henry Case, Chairman.....Phoenix
Mrs. Edward M. Hayden.....Tucson
Mrs. James H. Allen.....Prescott

Election of Officers
Report of Entertainment Committee
Report of Transportation Committee

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FRIDAY MORNING, MAY NINTH

10:00 A. M.—General Session

Greetings.....Mrs. C. A. Thomas, Tucson

Business Session:
Roll Call
Reading of Minutes
Memorial Service—
Mrs. W. Claude Davis.....Tucson
Miscellaneous Business—Resolutions
Installation of Officers

Speaker.....Dr. George T. Pack, New York City
Subject to be announced.

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ENTERTAINMENT

Thursday, May 8

Dinner.....7:00 P. M. Hotel Pioneer

Friday, May 9

Luncheon
In honor of Mrs. Jesse D. Hamer, President of the Auxiliary to the American Medical Association.

EVENING ENTERTAINMENT

Mrs. Dan L. Mahoney, Chairman

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PRESIDENTS AND SECRETARIES OF THE ASSOCIATION SINCE ITS ORGANIZATION

Year	Elected President	Secretary
1892	J. A. Miller, Phoenix	J. W. Green, Tucson
1893	H. A. Hughes, Phoenix	L. D. Dameron, Phoenix
1894	Ancil Martin, Phoenix	L. D. Dameron
1895	P. G. Cotter, Yuma	L. D. Dameron
1896	D. M. Purman, Phoenix	L. D. Dameron
1897	Chas. H. Jones, Tempe	O. E. Plath, Phoenix
1898	W. V. Whitmore, Tucson	O. E. Plath
1899	Wm Wylie, Phoenix	Chas. H. Jones, Tempe
1900	T. B. Davis, Prescott	Chas. H. Jones
1901	H. W. Fenner, Tucson	Chas. H. Jones
1902	Wm. Duffield, Phoenix	Chas. H. Jones
1903	L. D. Dameron, Phoenix	John W. Foss, Phoenix
1904	W. H. Ward, Phoenix	John W. Foss
1905	J. W. Coleman, Jerome	John W. Foss
1906	O. E. Plath, Phoenix	John W. Foss
1907	A. R. Hickman, Douglas	John W. Foss
1908	A. W. Olcott, Tucson	John W. Flinn, Prescott
1909	R. N. Looney, Prescott	John W. Flinn
1910	John W. Foss, Phoenix	John W. Flinn
1911	Francis E. Shine, Bisbee	John W. Flinn
1912	John E. Bacon, Miami	W. W. Watkins, Phoenix
1913	Ira E. Huffman, Tucson	C. E. Yount, Prescott
1914	John W Flinn, Prescott	C. E. Yount
1915	Roy E. Thomas, Phoenix	C. E. Yount
1916	Robt. Ferguson, Bisbee	C. E. Yount
1917	W. A. Holt, Globe	C. E. Yount
1918	W. Warner Watkins, Phoenix	C. E. Yount
1919	C. E. Yount, Prescott	D. F. Harbridge*, Phoenix
1920	A. M. Tuthill, Morenci	D. F. Harbridge
1921	A. L. Gustetter, Nogales	D. F. Harbridge
1922	H. T. Southworth, Prescott	D. F. Harbridge
1923	C. A. Thomas, Tucson	D. F. Harbridge
1924	R. D. Kennedy, Globe	D. F. Harbridge
1925	R. D. Kennedy**, Globe	D. F. Harbridge
1926	Geo. A. Bridge, Bisbee	D. F. Harbridge
1927	Chas. S. Vivian, Proenix	D. F. Harbridge
1928	A. C. Carlson, Jerome	D. F. Harbridge
1929	Samuel H. Watson, Tucson	D. F. Harbridge
1930	Joseph M. Greer, Phoenix	D. F. Harbridge
1931	Harry A. Reese, Yuma	D. F. Harbridge
1932	Clarence Gunter, Globe	D. F. Harbridge
1933	N. C. Bledsoe, Tucson	D. F. Harbridge
1934	Fred G. Holmes***, Phoenix Meade Clyne, Tucson	D. F. Harbridge
1935	C. R. K. Swetnam, Prescott	D. F. Harbridge
1936	Jesse D. Hamer, Phoenix	D. F. Harbridge
1937	C. R. Swackhamer, Superior	D. F. Harbridge
1938	Hal W. Rice, Bisbee	D. F. Harbridge
1939	Chas. S. Smith, Nogales	L. R. Kober, Phoenix
1940	D. F. Harbridge, Pheonix	W. W. Watkins, Phoenix
1941	W. Paul Holbrook, Tucson	W. W. Watkins
1942	E. Payne Palmer, Sr., Phoenix	W. W. Watkins and
1943	O. E. Utzinger, Ray	Frank J. Milloy, Phoenix
1944	Dan L. Mahoney, Tucson	Frank J. Milloy
1945	Charles P. Austin, Morenci	Frank J. Milloy
1946	George O. Bassett, Prescott	Frank J. Milloy

*Dr. Harbridge served 22 years as secretary due to method of election and time of taking office changing and overlapping.

**In 1925 the By-Laws were changed to provide for the election of a President-Elect, to be elected one year and take office the following year. For this reason Dr. Kennedy served two years as president.

***Dr. Holmes, the President-Elect, was compelled to resign before he became President on account of illness, and the Council advanced the first Vice-President, Dr. Meade Clyne, to that office, and he became President at the 1934 meeting.

Terms extend from one annual meeting to the next rather than for calendar year.

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TECHNICAL EXHIBITS

Audio Development Company
Minneapolis, Minnesota

D-9 AUDIOMETER

Exhibiting the A D C Audiometer of real appeal to the Otologist and Pediatrician.

Ayerst, McKenna & Harrison.....**Booth No. 15**
New York City
Biological and Pharmaceutical Chemists.

Baby's Valet.....**Booth No. 11**

Baby's Valet

Tucson and Phoenix
Sanitized Diaper Service.

Don Baxter, Inc......**Booth No. 6**
Glendale, California

DON BAXTER, INC.
RESEARCH AND PRODUCTION LABORATORIES
1015 GRANDVIEW AVENUE
GLENDALE 1, CALIFORNIA

Latest developments in the parenteral-therapy field will be featured in the exhibit of Don Baxter, Inc., including the 7 Trinidex solution; Vacosets (Baxter's expendable administration sets), alcohol solutions, and latest blood-transfusion equipment and techniques.

Blair Surgical Supply.....**Booths No. 17-18-10**
Tucson and Phoenix offices

Surgical Equipment; X-ray Engineering; Laboratory Supplies; Rocky Mountain distributor for Picker X-ray Equipment.

Blue Cross Hospital Service.....**Booth No. 29**
Phoenix, Arizona

Arizona's Blue Cross Hospitalization Plan, non-profit and voluntary, now has 45,000 subscribers. A representative will be in attendance to provide literature and to answer questions relative to this popular hospitalization service.

Cameron Surgical Specialty Co......**Booth No. 22**
Chicago, Illinois

See the new Cameron Electro-Surgical units, Coagulair-Sigmoidoscope, Electro-Diagnostosets, Flexible Gastrascopes, Bronchoscopes, Esophogoscopes, Larymoscopes, Mirrolite, Binocular Spectacle Loupe, Magniscope and other specialties.

Ciba Pharmaceutical Products, Inc...**Booth No. 20**
Summit, New Jersey

Ciba Pharmaceutical Products, Inc., Summit, New Jersey, Booth No. 20, invite you to visit their booth for latest information on PYRIBENAMINE, the new anihistaminic and antiallergic compound. The representative in attendance will gladly answer any questions that you may have concerning this and other Ciba products.

Cutter Laboratories.....**Booth No. 16**
Berkeley, Calif.

At the Cutter Laboratories exhibit, a new Leur-like disposable syringe of Penicillin in Oil and Wax will be on display. You will also see the complete and exclusive line of Blood Fractions, including Albumin, Fibrin Foam, Hypertussis, Immune Serum and Globulin. Abstracts of the literature related to these products will be available.

The Doho Chemical Corporation
New York, Montreal, London

The makers of AURALGAN are introducing at this meeting their new sulfa drug preparation O-TOS-MO-SAN, indicated in the treatment and control of chronic suppuraive ears. Our representaeives will be happy to explain, in detail, the workings of these medications. We will also distribute our latest series of three Anatomico-Pathologic Charts of the Ear, in color suiable for framing.

C. B. Fleet, Inc......**Booth No. 12**
Lynchburg, Virginia

C. B. Fleet Co., Inc., cordially invites you to stop by Booth No. 12 for a short visit with Mr. Fred T. Bergstrom, the representative who sees you in your office about once a year. Perhaps there is something about Phospho-Soda (Fleet), the pure, stable aqueus concentrate of the two U.S.P. Solium Phosphates, you would like to discuss with him.

General Electric Corporation.....**Booth No. 4**
Phoenix, Arizona

Factual discussions with members of our sales and service organization during the state meeting will aid you in your future apparatus planning. Why not drop in and avail yourself of our wide experience and know-how?

Lanteen Medical Laboratories, Inc.
Chicago 10, Illinois

"Lanteen Medical Laboratories, Inc., Chicago 10, Illinois, cordially invite you to their exhibit of their well known pharmaceuttcal specialties. Included will be natural and synthetic estrogenic products, ESTROGEL and HEXYPHEEN, VI-TEENS products, including VI-TEENS HOMOGENIZED Vitamins, as well as their line of gynecic specialties, will also be included."

Lederle Laboratories, Inc.
New York City, N. Y.

A display of the latest products in the biological and pharmaceutical fields.

Maico Southwest.....**Booth No. 1**
Phoenix, Arizona

Precision audiometers. Representative will be present to demonstrate.

M & R Dietetic Laboratories, Inc.....**Booth No. 26**
Columbus, Ohio

M & R Dietetic, Laboratories, Inc., booth No. 26, will display Similac, a food for infants deprived either partially or entirely of breast milk. Messrs. M. R. Davis and Miles Engle will appreciate the opportunity to discuss the merit and suggested application for both the normal and special feeding cases.

Mead Johnson & Company.....**Booth No. 13**
Evansville, Indiana

Amigen and Protolysate will be on display at the Mead Johnson Exhibit at your Arizona State Medical Meeting. Mead Johnson has pioneered the amino acid field commercially; the products have been described in more than one hundred and forty articles in the medical literature; this year they are available. Trained representatives will be at the Mead Exhibit to discuss details of the new amino acid products. Shown also will be Dextri-Maltose, Pablum, Pabena, Oleum Percomorphum and the other Mead prolucts used in Infant Nutrition.

Ortho Pharmaceutical Corporation
Raritan, New Jersey

Ortho Pharmaceutical Corporation extends a cordial invitation to visit Booth No. 24, where they will exhibit their complete line of Gynecic Pharmaceuticals. Samples and literature will be available upon request.

Pet Milk Corporation.....**Booth No. 10**
St. Louis, Missouri

There will be a complete display of material illustrating the time-saving Pet Milk services available to physicians. The trained representatives in attendance invite you for a visit to give you information about the use of Pet Milk for infant feeding. Miniature cans will be given to physicians visiting the exhibit.

A. H. Robins Company.....**Booth No. 23**
Richmond 19, Virginia

"Ethical Pharmaceuticals of Merit Since 1878."

Southwestern Surgical Supply Co...**Booths 7 & 8**
Phoenix, Arizona

Serving the medical profession for 25 years.

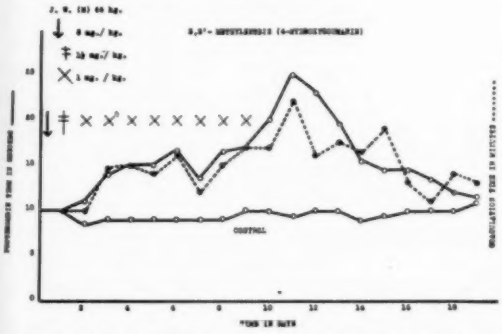
Standard Surgical & Hospital Supply Company
Phoenix, Arizona **Booths No. 2 & 3**

We will have on display latest X-ray diagnostic equipment manufactured by North American Phillips Company, together with the latest type of Physio-Therapy equipment. We will also display a complete line of surgical instruments and physicians' equipment and supplies.

THE EXPERIMENTAL AND CLINICAL EFFECT OF DICUMAROL ADMINISTRATION

Early in our studies[18] it was demonstrated in animals that Dicumarol did not have great toxicity in dogs and that the liver was not damaged even by excessive doses of the drug. Hemorrhage, however, which was often widespread and usually involved the gastro-intestinal tract, was a common result of excessive dosage and was frequently attended by widespread dilatation of the small blood vessels. It was possible to demonstrate that of the dogs given a single dose of 5 mgs./Kg. of Dicumarol, a much smaller proportion than of the control dogs developed venous thrombosis. Thrombosis was induced in the radial veins by the injection of the sclerosing agent monolethanolamine oleate (monolate); with the technique used, 50 per cent of the control animals developed thromboses but only 6 per cent of the Dicumarol-treated dogs[19].

Figure 1 shows the effects of Dicumarol upon the prothrombin time and coagulation time in one human being studied. Studies of liver function, renal function, erythrocyte, leukocyte, and platelet counts were all negative.



The effect of Dicumarol upon the prothrombin time and coagulation time in a 66 kg. white male. The line marked control represents the prothrombin times of an untreated patient picked at random on the ward each day and offers check upon the potency of the thromboplastin employed and the technique of the prothrombin time test.

The indications for anticoagulant therapy are many. Thus an anticogulant may be wisely used prophylactically in operations where the incidence of thrombosis is high, as in surgery of the pelvis, vascular surgery and after splenectomy. Anticoagulant therapy is especially indicated for patients who at the time of previous surgery suffered thrombophlebitis or embolism. These drugs are indicated when there is thrombophlebitis and phlebothrombosis with or without pulmonary infarction, unquestioned cerebral thrombosis, mesenteric thrombosis, and probably coronary thrombosis.

Thrombophlebitis and phleborthrombosis are very common. The latter, which is especially likely to produce subsequent pulmonary embolism, often occurs without pain and is not infrequently overlooked. Many methods of treating these conditions have been suggested in recent years. Ligation of the femoral vein, or iliac vein, as advised by Homans[20] (some surgeons have advocated bilateral ligation) is perhaps the surest method of preventing pulmonary embolus and infarction. It is not always a simple surgical procedure and, in the opinion of some, is rather drastic. The need for it increases with the advancing years[21], since the incidence of serious and fatal embolus increases with each decade beyond forty. OOchsner and De Bakey[22] have advocated regional sympathetic ganglion block, previously advocated by Leriche to relieve the arterial and venous spasm and pain. It is presumed that the blood flow is thus speeded up and the development of thrombus minimized.

It need hardly be said that anticoagulant therapy will not resolve a thrombus already formed, whether it is associated with inflammation of the vein or not. If phlebitis exists, the thrombus usually adheres to the vein and is unlikely to travel. The purpose then of anticoagulant therapy is to prevent the development of a second thrombus elsewhere, or a propagated thrombus — a fresh thrombus at the site of the original one — a thrombus which is likely to be loosely attached, and readily broken off to form an embolus. This is the logic of anticoagulant therapy in these cases. And since Dicumarol does not exert its effect for 24 to 48 hours, the ideal therapy is the early institution of heparin administration with concomitant exhibition of Dicumarol. As soon as the prothrombin time is prolonged, one may assume that the Dicumarol is exerting its effect and that the heparin may be dispensed with. Some caution is necessary, however, since large doses of heparin produce hypoprothrombinemia, and on rare occasions, ordinary doses of heparin may cause rduction in the prothrombin.

The results of this type of therapy in the hands of capable physicians are best exemplified in the large series of clinical cases at the Mayo clinic, recorded in the paper of Barker in the August, 1946, issue of Minnesota Medicine[23]. Some of the results, which are truly impressive, are shown in

the following two tables, which Doctor Barker has kindly permitted me to use.

TABLE I

NONFATAL POSTOPERATIVE PULMONARY EMBOLISM AND INFARCTION

	Control group; anticoagulants not administered		Dicumarol administered	
	Number	Per Cent	Number	Per Cent
Total	678	100	292	100
Subsequent venous thrombosis, pulmonary embolism or infarction	297	43.8	3	1.0
Subsequent fatal pulmonary embolism	124	18.3	1*	0.3

*After prothrombin had returned to normal.

TABLE II

POSTOPERATIVE THROMBOPHLEBITIS

	Control group; anticoagulants not administered		Dicumarol administered	
	Number	Per Cent	Number	Per Cent
Total cases	897	100	280	100
Subsequent venous thrombosis, pulmonary embolism or infarction	227	25.3	8*	2.8
Subsequent fatal pulmonary embolism	51	5.7	0	0

*One case of minor pulmonary infarction and seven cases of thrombophlebitis in three of which the value for prothrombin was greater than 30 per cent when the reccurrence developed.

Our findings in a much smaller series of cases of thrombophlebitis and pulmonary embolism tend to confirm Barker's observation[23] that subsequent thrombophlebitis and pulmonary embolsm rarely occur after anticoagulant therapy has been properly administered.

The one hazard is hemorrhage, which may be serious and difficult to control. The best insurance against this, I believe, is accurate daily determination of the prothrombin time. In a hospital this determination should be done by a single technician, one who is well trained in the procedure and one who does these tests frequently. In our experience, repeated several times now, that the best of technicians must spend a month in doing daily determinations to acquire the skill and accuracy required. Once trained, she should handle all the patients treated with Dicumarol. Usually those who have had trouble with the drug have been relying upon inaccurate determinations of prothrombin time and thus have had a false sense of security.

I have attempted this morning to outline for you what is known about anticoagulants at the present time, to cite some of the indications and contraindications for their use, and to describe some of the results that may be obtained form their proper employment. The use of both heparin and Dicumarol involves no special hazards except that of gross hemorrhage from overdosage or, in the unusually susceptible individual, from the usual dosage. The disadvantages of heparin are that it costs much more and requires parenteral administration, whereas Dicumarol is cheap and may be given by mouth. When heparin is employed the coagulation time must be determined; when Dicumarol is used, the prothrombin time, a technically more difficult, but equally accurate, determination when properly done, must be measured. Heparin has a very real advantage in that the coagulation time of the blood will return to normal within a few hours after administration has been stopped, thus permitting much better control of the theapy.

Finally, I would emphasize the need for employing anticoagulants when they are indicated and only when they are indicated, careful individualization of therapy, and close daily attention to the patient while under therapy. The anticoagulants are useful but dangerous drugs; almost never should they be employed in patients who are not hospitalized.

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H. INFLUENZAE MENINGITIS
(with Report of Incidence in a Newborn with Recovery)

MILTON C. F. SEMOFF, M.M., F.A.C.P.
Tucson, Arizona

MENINGITIS caused by H. Influenzae, is of great interest to the pediatrician, as a matter of pride in the lowered mortality, with deference to the incidence in children (ranking first to third), and with humility in the difficulty of early recognition of the indicative signs and symptoms.

The lowered mortality can be seen in the report from the Hospital for Sick Children, Toronto. They reported 166 cases during 1919 and 1944. The early group of 70 cases had a 98% fatality. The next group of 50 cases using horse and guinea pig sera had a 78% fatality. The next group of 20 patients introduced the early sulfonamides and above sera with a reduction to 75% fatality. The most recent group of 16 patients treated with anti-B rabbit sera and usually sulfadiazine reduced the fatality to 50%; and the last group of 10 patients treated with sulfadiazine alone, also reported a fatality of 50%. I surveyed nine cases at Cridren's Hospital in Chattanooga from 1940 to 1942; we had four recoveries out of nine cases treated with varying sulfa drugs.

Influenzae Meningitis is more common in children than is usually considered; varying with the incidence of epidemics and the locality concerned, it ranks first and never lower than third, in all forms of meningitis; meningicoccoc and pneumococcoc meningitis, being the other two common forms encountered.

The definite diagnosis of H. Influenzae meningitis is made bacteriologically from spinal taps, blood cultures, and nasopharyngial smears. However, it is early recognition of indicative signs and symptoms of meningitis leading to the above procedures that is of major importance. During the first year of life, any unexplained fever associated with irritability, alternating with drowziness, a high pitched cry, and glassy eyes, should warrant a spinal tap. After the first year, any unexplained fever with or without drowziness, transient delirium, hyperactive reflexes, generalized hyperaesthesia, resistance and pain on neck flexion, positive Kernig and Brudjinski reflexes, should likewise lead to spinal puncture. Buldging fontanelles, stiff neck, and opisthotonus are all *too late* signs.

Reported at the Staff Meeting, Tucson Medical Center, Arizona.

Bacteriologically H. Influenzae is a gram negative small coccobacilli with filamentous forms, and it stains poorly. If the organism is obtained directly, a positive diagnosis can be made by demonstration of capsular swelling. This is done in exactly the same manner as pneumococcoc typing, with type specific sera, swelling of the capsule, and usually agglutination present in a few minutes. However, no typing should be considered negative until thirty minutes have elapsed. If no organism is demonstrable, cultures must be made. Alexander uses Leventhal broth agar plates and blood agar plates. Within twelve to twenty four hours, small colonies will be available for typing. If there is a mixed flora, two or three drops of a penicillin solution (ten units per milliliter) on a chocolate agar plate with heavy inoculation will inhibit all organisms except H. Influenzae. Inoculation at the same time of an agar slant will allow the other organisms to grow readily while H. Influenzae grows poorly. Within twelve hours the organism will be available for typing. It should be remembered that the rough forms will not type, but these are seldom pathological.

The therapy for this disease can be roughly considered as the sulfonamides, Anti-B Influenzae rabbit serum, penicillin and streptomycin. Almost all of the sulfonamides have been reported favorable at some time. The consensus of opinion at the present favors sulfathiozole; this is probably due to the accessibility of the drug. Type B is the most common group of pathologic H. Influenzae. Of seventy one cultures reported at Toronto seventy were type B. Rabbit sera is available only for this type. Penicillin as shown by many investigators does not inbibit the growth of H. Influenzae. However, many reports are available showing clinically "a benifical synergy." Streptomycin has been reported very favorably but is not as yet generally available.

The following outline of therapy is the sum total of various investigators reports. However, basically, they all follow Alexander's work. When an impression of meningitis has been made, a spinal tap should be done, blood cultures should be taken and if feasible, nasopharyngial smears and cultures should be made. Upon com-

pletion of the above procedure, sodium sulfadiazine should be given subcutaneouly (.1 gram per kilo).

This is recommended even if the drug can be tolerated by mouth, in order quickly to build up a high level. Adequate fluids are administered in the interval of waiting for the laboratory reports. Penicillin or streptomycin may also be given at this time. If the organism is a type B and the clinical condition warrants Antibody. it may be administered intravenously. This is more efficient if administered after twelve hour delay, except in chronic or severe cases.

Sulfadiazine alone, is recommended if the patient is seen within twenty-four hours of the onset, if the spinal fluid is forty or above, or if after forty-eight hours the spinal fluid is sterile, and the clinical condition is improved. The drug is continued for two weeks after the spinal fluid is setrile and normal in its constituents. It has been recommended that the original subcutaneous dose be repeated at twelve hours for at least two doses. Orally, sulfadiazine should be administered with sodium bicarbonate (two to three times the amount of sulfa) in an attempt to maintain a urine pH of 7.5. Extra fluids are given by the route the patient can most easily tolerate and assimilate. For intravenous use .6 M Lactate with 5% glucose at the rate of twenty c.c. per kilo per hour for the first two hours, has been recommended. A blood level of 15 mgm% is desirable.

Type B Anti H. Influenzae serum is to be administered in all the chronic or severe cases and those which have not responded favorably to the sulfonamides. The most desirable procedure is to type the organism first. If sera is to be given, the amount is calculated in the vials of twenty-five mgm antibody nitrogen.

This is based upon the spinal fluid sugar expressed in mgm%. The necessary chart accompanies each ampoule. Enough antibody should be given in the first dose if possible to supply a surplus in the blood stream, so that it will not be necessary to repeat. The sera is diluted with ten c.c. of normal saline per kilo and adjusted to drip for two hours. If possible, this should be followed in twelve to twenty-four hours with a test for sufficiency of antibody. The patient's blood is drawn; the serum is diluted to ten and tested against a known organism for swelling. In severe cases more antibody must be administered and daily controls taken.

For the very severe cases in which there has been no favorable response Alexander recommended intra-thecal administration of the antibody. This is definitely a last stand procedure. She has reported a very interesting procedure in intra-thecal block: one c.c. of heparin is first given intra-thecally to clear the block; this is then followed by the sera.

Penicillin has been referred to previously. Three recoveries were reported in Uraguay with the use of sulfathiazole and penicillin. The authors compared their results with the previous poor reports of sulfathiazole alone. They feel definitely that the penicillin was the difference. A type F.H. Influenzae meningitis with recovery was reported using sulfathiazole, sulfadiazine, and penicillin. The author here, also offers his opinion of a definite "beneficial synergy." My case which recovered, was given sulfadiazine and five thousand units of penicillin every three hours for seven days.

In treating H. Influenzae Meningitis, spinal punctures must be done almost daily until the fluid is clear and the chemistry and cell count approach normal. This differs from the accepted treatment of the other common forms of meningitis. There should be no fear of doing these taps as frequently as necessary. One report from Los Angeles County Hospital told of a two year old female child that had sixty-nine taps in seventy-five days with recovery and residual findings. Case No. 451A T.M.C.

D. B. white male was born on June 7, 1945, at the Tucson Medical Center, weight 7 pounds 9 ounces, height 19½ inches. The mother was a multapara, who had severe vomiting for the first three months of pregnancy and at term developed a mild toxemia (urinary albumin, blood pressure 190mm). She was induced by rupturing of the membranes and had an uneventful delivery. The baby crie dlustily. He was put to the breast with lactose water after feedings. His weight fell to 6 pounds 15 ounces on the fourth day but by the sixth day he had gained to 7 pounds. Early on the morning of the sixth day his temperature rose to 101.4 F; by 8:00 A.M. the nurse noted "a sharp shrill cry, as if in pain." He refused to nurse and the obstetrician ordered a formula. The baby would fall asleep and then awake fretful. On the seventh day the temperature remained elevated, rising to 102.2 F at 5 P.M. The nurses noted "eyes blurry, respiration difficult at times, fretful, and crying while feed-

ing." I first saw the child at 5 P.M. and noted a mild jaundice, mild spasticity of the extremities irritable, sharp cry, and mild dehydration.

He was given calmulsion by mouth and Ringer's Solution subcutaneously. The baby had a quiet night, took his feedings well, but the temperature spiked to 104 F. The next morning, the eighth day, forty-eight hours since the first symptom, the infant looked better, but a spinal tap was done. The CNS fluid was under no marked pressure, xanthrochromic, and cloudy. Penicillin was started by hypo and sulfadiazine by mouth (one grain per pound stat and two grains per pound per twenty-four hours). Spinal fluid cell count 40,600; 85% polys, 15% lymphs; smear showed diplobacilli. The next day culture was identified as H. Influenzae. Bloodcount showed; Hb. 13.2 gms, RBC 4,560,000 WBC 16,-550 Segs 71%, Stabs 17%, lymphs 11%. Eosin 1%. That night the baby had a severe cyanotic spell, responded to Cormine, and then continued to take the medication, water, and formula by mouth. On the tenth day, for the first time, neck became stiff with mild oposthotonus and cyanosis. Oxygen was given PRN. On the twelfth day, the baby had severe cyanotic spells with convulsions and muscular twitching. On the thirteenth days, the fontanelle was full. However, the temperature remained under 100 after this date. The infant oral feeding and medication. Cisterna puncture gave relief after the dry spinal taps. Mild convulsions at night with Syanosis persisted until the ninteenth day; thereafter, the clinical course was upgrade. On the thirty-ninth day he weighed 9 pounds, the CNS fluid had twenty-two cells, and the culture negative. He was discharged on the forty-fifth day weighing 9 pounds 11 ounces, feeding well, active, with no evident residual findings.

He was seen in the office at six and a half months old weighing 17 pounds, 12 ounces, happy, active, feeding well. At one year and three months of age he weighed twenty-two pounds, was thirty-one inches tall, and showed no residual findings of his menningitis.

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PRECANCEROUS DERMATOSES

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PRECANCEROUS dermatoses are skin lesions which may undergo malignant change. Some of them regularly and invariably become cancerous, namely erythroplasia and xeroderma pigmentosum. Others, such as senile keratoses frequently become malignant, but do not do so invariably. Still others, such as secaceous cysts, rarely become cancerous. In this short paper each condition will be discussed in such detail as its importance would seem to warrant.

The senile keratosis occurs as a reddish or reddish-brown scaly spot varying in size up to one centimeter or more in diameter. It is irregular in outline and usually little if any raised above the surface of the surrounding skin. The favored areas are those most frequently exposed to the sunlight, namely the dorsal surfaces of the hands and forearms, and the face. The patient usually is of light complexion. Treatment consists of removing the lesion. This can be done by electrodesiccation, which probably is the simplest, or by x-ray or radium therapy, the hot cautery, carbon dioxide snow, or by cold scalpel surgery. With suitable treatment, complete cure of the lesion is assured.

Presented before the Maricopa County Medical Society, October 7, 1946.

Several other conditions are similar to senile keratoses, namely chronic actinic dermatitis, chronic radiodermatitis, xeroderma pigmentosum, and cornu cutaneum. Chronic actinic dermatitis is known more familiarly as sailor's skin or farmer's skin. It usually occurs in middle-aged or elderly persons, (rarely in the young) who are fair-complexioned, often red-haired and freckled, and who have led an outdoor life with long exposure to sunshine. In this condition one finds very numerous typical keratoses, in addition to dryness, wrinkling, freckling, and telangiectasia. Chronic radiodermatitis is much the same as chronic actinic dermatitis except that it is usually localized to a smaller area. Xeroderma pigmentosum closely resembles chronic actinic dermatitis, but begins in early childhood, and is considered to be concerned with a congenital hypersensitivity to sunlight. Keratoses or carcinomas that appear in chronic actinic dermatitis, chronic radiodermatitis, or xeroderma pigmentosum must be removed by appropriate methods. Cornu cutaneum, which is another simple hyperkeratosis, should be removed with destruction of its bse to prevent cancer, although it must be stated that cancer resulting from a cutaneous horn is not common.

The seborrheic keratosis is of little importance as a precancerous lesion, inasmuch as malignant degeneration rarely occurs. It is the yellowish to brownish-black lesion seen most frequently on the seborrheic areas of the back, but also sometimes on the chest and face, and elsewere. It is quite flat at first, later becoming thicker and darker sometimes even taking on a verrucous appearance. It varies in size up to about two centimeters in diameter, and is round or oval in shape and shows a sharply demarcated border. Frequently the patient requests removal of this lesion either for cosmetic reasons or because of itching.

Certain chemical substances produce keratoses. Many of these result from occupational exposures. Some of the chemical agents known to be serious offenders are arsenic, carbon, hydrocarbons, and products such as soot, aniline, pitch, paraffin, tar, anthracene and petroleum. Arsenical keratoses, it must be pointed out, occur in persons who have received arsenic as medication as well as in those who have had occupational exposure. The arsenical keratosis shows a predilection for the palms and soles, a fact which renders the mdistinguishable in many cases from other keratoses. The workers who develop these various chemical keratoses because of occupational hazards include sailors and fishermen, tailors, textile workers, railway engineers and firemen, gas works employees, petroleum workers, tar and paraffin workers, chimney sweeps and many others.

There are two conditions which are brought up at this point, not because they are classified as keratoses, but rather as dyskeratoses, namely Bowon's Disease and Paget's disease. These two conditions are merely to be mentioned and then dismissed, not because they are unimportant, but rather because they are no longer considered to be precancerous lesion. They are cancers from their inception.

Nevi or moles occur very frequently, but malignant melanomas although they are frequently seen, must occur in only a small percentage of moles. Certain types of nevi seem to be more frequently related to malignancy than others, namely the very black or blue-black nevi. These lesions occur most frequently on or about the face, but may occur at other sites, especially about the feet and toes. The brown pale nevi, especially those possessing hairs, are usually not malignant. Most authorities consider it unwise to fulgurate nevi, or to use acids or refrigeration in an attempt to destroy them. Radiation therapy is not successful. The hot cautery for local destruction or scalpel considered good and safe methods. The chances of recovery are slight once a malignant nevus has started to grow, especially if metastases have already occurred.

Precancerous lesions of the mucous membranes include the common leukoplakia, the less common kraurosis vulvae, and the uncommon erythroplasia.

Leukoplakia occurs on all parts of the mouth, including the tongue, the lips, the gums and the tonsils as well as the cheeks. It also may be seen occasionally on the penis and about the female genitalia. The mouth lesions are found predominantly in men, only 5% of the patients being women. The lesion appears as a white patch, variable in size, usually smooth or only slightly rough. Symptoms are usually absent or negligible. Syphilis is a predisposing factor in oral leukoplakia, as is poor oral hygiene. Almost all of its victims are smokers.

Inasmuch as one patient in every three suffering from leukoplakia, develops squamous-cell carcinoma, the lesions must be kept under close

observation, and oral hygiene must be perfect. Any lesion showing suspicious changes should be excised or destroyed with the electrocautery. Radiation therapy is not advised because recurrence is common following this procedure.

Kraurosis vulvae is a relatively uncommon condition found in elderly women, beginning as red spots about the vaginal orifice which extend to involve large areas and later become pale, atrophic and sclerotic with ultimate retraction of the tissues. The cause of the condition is not known, but it is certain that degenerative changes associated with age, and chronic irritation both may play a part. Treatment may be conservtive if the patient is kept under frequent observation. Hormonal therapy is advocated by some investigators. If a suspicious change in the lesion should appear, vulvectomy may be indicated.

Erythroplasia is an uncommon condition involving the glans and prepuce or, less frequently, the vulva, and still less frequently, the mouth. The lesions are red patches varying in size up to a diameter of one centimeter or more. They may be dry or moist, with a shiny or velvety appearance. Carcinoma appears ultimately in all untreated cases, and all except radical treatment is unsuccessful. Surgical removal of the lesion is advocated once the diagnosis is established by biopsy.

There is a miscellaneous group of skin disorders which infrequently undergo malignant change. Little discussion of these conditions is necessary to this paper. Old scars, especially those due to burns, develop carcinoma often enough to cuse one to be on his guard. The same is true of ulcers of long standing, especially when infection is present. Syphilis, especiallly in the mouth, more especially in conjunction with leukoplakia, and most especially on the tongue as interstitial glossitis, is a frequent offender. Finally discoid lupus erythematosus, lupus vulgaris, sebaceous cysts, Von Recklinghausen's neurofibromatosis, and other skin disorders may at times develop cancers. All of these are to be managed in a manner indicated by the prevailing circumstances.

SUMMARY

The precancerous dermatoses are discussed briefly. Emphasis is placed on diagnosis, treatment, and prognosis.

STUDIES ON THE EFFECT OF DILUTING FLUIDS ON SEROLOGICAL TITRES

II. A Comparison of the Titres Obtained When the Sera Are Diluted Prior to Inactivation and When They Are Diluted After Inactivation

EDWARD L. BREAZEALE[1], THEODORE R. REUSSER[2], and L. F. PIERCE[3]

THIS is the second of a series of papers deallining with the subject of serological titering of Studes on the effect of diluting fluids on . . . syphilitic sera. In an earlier paper[1] the effect of diluting with (a) normal saline (b) 2.5% saline and (c) seronegative sera was discussed.

If we assume that seropositive blood is reactive due to the presence of an antibody-like substance, which we may call reagin, it would appear that the titre should not be affected by the type of diluting fluid used. However, in the previous paper dealing with this subject, the authors brought out the fact that by using pooled seronegative sera instead of saline, a titre of from four to ten times as great could be obtained. It is of interest to note that in "Technics of Serodiagnostic Tests for Syphilis, 1944"[2] that several of the author serologists recommend normal saline (Kahn, Wassermann, Kline, Mazzini, Eagle), some recommed 2.5% saline (Kahn), while still others prefer the use of pooled negative sera (Kline, Hinton). From this it will be seen that if a serum was diluted with saline and titered by the method of Mezzini or Eagle in one laboratory, and then another laboratory titered the same sample by the method of Kline or Hinton using pooled negative sera, there would be an apparent discrepancy in results.

EXPERIMENTAL

In an effort to help explain the observed phenomenon reported in the first of these papers

1. Arizona State Board of Health—Division of Laboratories, Tucson, Arizona.
2. Tucson Medical Laboratories, Tucson, Arizona.
3. L. F. Pierce Laboratories, Los Angeles, California.

the following experiments were carried out in this laboratory.

Blood samples, as they were received by this laboratory, were sparated from their clots, and centrifuged at high speed for five minutes and the clear supernatent sera poured off. The sera were then divided into two portions, one "inactived" in an electrically controlled water bath at 56° C for 30 minutes, and 4) raw sera diluted at 56° C for a period of 30 minutes and the seeand portion used for the raw dilution studies. The inactivated samples were then examined according to the methods of Kahn, Kline and Hinton. Serial dilutions were then made as follows: 1. Inactivated esra diluted with 2.5% saline; 2. inactivated sera diluted with inactivated seronegative sera; 3. raw sera (second portion above) diluted with saline and then inactivated at 56° C for 30 minutes, and 4. raw sera diluted with raw seronegative sera and then inactivated at 56° C for 30 minutes. All dilutions were examined by the same technics as were the original samples. Only those results obtained by the Kahn technic are reported in Table I. Standard Klines and Rapid Rintons were run on each dilution, while only a one tube Kahn (0.025 ml antigen as recommended by Kahn) was used in determining the titre.

RESULTS

TABLE I (See page 4)

Individual readings on representative samples are given in Table II.

TABLE II (See page 4)

DISCUSSION

The results indicate that (a) a higher titre is obtained when sera is used as a diluting media than those obtained where saline is employed and (b) that a higher titre is obtained when the sera is diluted raw and then inactivated than when inactivated sera is titred.

There is no readily available explanation for this observation. Obviously, the basic mechanism responsible for the production of a positive serological test must be linked in some way to a thermo-lable substance in the sera. It would appear that there must be some type of molecule

TABLE I

THE EFFECT ON TITRE OF DILUTING SERA BEFORE AND AFTER INACTIVATION
(Kahn Results[1])
Number remaining positive

Diluted with	DILUTIONS 1:1	1:2	1:4	1:8	1:16	1:32	1:64	1:128	1:256	1:512	1:1024	1:2048
Control Reaction 4+ (36 Sera)												
Saline Raw	36	36	35	29	16	6	3	1	0	0	0	0
Sera Raw	36	36	36	36	34	27	20	10	3	3	2	0
Saline Heated	36	35	27	14	7	2	0	0	0	0	0	0
Sera Heated	36	36	36	29	22	11	6	1	0	0	0	0
Control Reaction 3+ (18 Sera)												
Saline Raw	18	17	14	7	3	0	0	0	0	0	0	0
Sera Raw	18	18	18	17	12	8	3	0	0	0	0	0
Saline Heated	18	12	5	0	0	0	0	0	0	0	0	0
Sera Heated	18	17	15	5	4	0	0	0	0	0	0	0
Control Reaction 2+ (12 Sera)												
Saline Raw	12	12	9	1	0	0	0	0	0	0	0	0
Sera Raw	12	12	12	10	6	1	0	0	0	0	0	0
Saline Heated	12	10	1	0	0	0	0	0	0	0	0	0
Sera Heated	12	11	5	0	0	0	0	0	0	0	0	0

Control Reaction 1+ (6 Sera)												
Saline Raw	6	5	4	1	0	0	0	0	0	0	0	0
Sera Raw	6	6	6	1	0	0	0	0	0	0	0	0
Saline Heated	6	4	2	0	0	0	0	0	0	0	0	0
Sera Heated	0	5	2	0	0	0	0	0	0	0	0	0
Control Reaction Negative (25 Sera)												
Saline Raw	0	0	0	0	0	0	0	0	0	0	0	0
Sera Raw	0	0	0	0	0	0	0	0	0	0	0	0
Saline Heated	0	0	0	0	0	0	0	0	0	0	0	0
Sera Heated	0	0	0	0	0	0	0	0	0	0	0	0

1. Complete agreement with Kline, Hinton results.

TABLE II

INDIVIDUAL DILUTION READINGS COMPARING THE EFFECT OF DILUTION MEDIAS
(Kahn Results[1])

Sample No.	Control Reaction	Diluent	DILUTIONS 1:1	1:2	1:4	1:8	1:16	1:32	1:64	1:128	1:256	1:512	1:1024
1	4+	Saline Raw	4+	4+	4+	3+	2+	1+	—	—	—	—	—
		Sera Raw	4+	4+	4+	4+	4+	3+	3+	2+	2+	1+	+
		Saline Inact.	4+	4+	3+	2+	1+	—	—	—	—	—	—
		Sera Inact.	4+	4+	4+	3+	2+	1+	+	—	—	—	—
30	3+	Saline Raw	3+	2+	1+	+	—	—	—	—	—	—	—
		Sera Raw	4+	3+	3+	2+	1+	+	—	—	—	—	—
		Saline Inact.	2+	1+	—	—	—	—	—	—	—	—	—
		Sera Inact.	3+	2+	1+	—	—	—	—	—	—	—	—
25	2+	Saline Raw	2+	1+	—	—	—	—	—	—	—	—	—
		Sera Raw	3+	3+	2+	1+	—	—	—	—	—	—	—
		Saline Inact.	1+	1+	—	—	—	—	—	—	—	—	—
		Sera Inact.	3+	3+	2+	—	—	—	—	—	—	—	—
18	1+	Saline Raw	1+	1+	+	—	—	—	—	—	—	—	—
		Sera Raw	3+	2+	1+	—	—	—	—	—	—	—	—
		Saline Inact.	1+	+	—	—	—	—	—	—	—	—	—
		Sera Inact.	2+	1+	—	—	—	—	—	—	—	—	—
97	Neg.	Saline Raw	—	—	—	—	—	—	—	—	—	—	—
		Sera Raw	—	—	—	—	—	—	—	—	—	—	—
		Saline Inact.	—	—	—	—	—	—	—	—	—	—	—
		Sera Inact.	—	—	—	—	—	—	—	—	—	—	—

1. Complete agreement with Kline and Hinton results.

splitting substance in a seropositive sera that is thermo-lable. If this were true, we could have some logical basis for inactivation of sera for flocculation precipitation tests. It is a well known fact that unless a serum is strongly positive all of the conventional tests will show negative results on a raw sample. This same phenomenon is probably responsible for the increased titre when raw sera are diluted with pooled negative sera. From the results obtained on a small series no difference was observed when raw negative sera or inactivated negative sera were used for a diluent. This fact would indicate that the fraction responsible for these increased titres was elements, in the positive sera is activating a portion of the negative sera, thereby raising the

titre. This element is probably not completely destroyed by heat, for if it were, it would be expected that the same titre would be obtained when saline was used as when negative sera were used as a diluting fluid.

It is of interest to note from Table I that approximately the same titres were obtained when raw sera were diluted with saline as when heated sera were diluted with negative sera. Here again no readily available explanation is available, and one can only offer a hypothesis. It appears probable that in the case of the raw sera and the saline that we are dealing with an ionic concentration factor, that is that the sera will come to a constant ionic concentration to any given dilution. When that seru mis diluted, the ionic concentration, (Ca)++, becomes lowered and thereby permitting additional (Ca)++ to become ionized. The vector causing this ionization is still highly active since the sample has not been "inactivated" by heat. On the other hand if the force, or vector, causing this ionization has been destroyed or inhibited by heating, and then the heated sera diluted with saline a simple diluting out of the active particles results and a negative test is obtained. However, apparently this active force, or vector, is not completely destroyed by heat and when a sera is titred with negative sera it exerts some degree of force on that diluent and thereby increases the ionization of the salts and consequently the titre.

These explanations are offered only as an hypothesis. Work is continuing in this laboratory and it is hoped a more complete explanation will be forthcoming shortly.

CONCLUSION

1. Higher titres are obtained when a sera is titrated before inactivation than is obtained on heated sera regardless of whether sera or saline is used as the diluent.

2. The same titre is obtained if raw pooled negative sera is used as where inactivated pooled negative sera is the diluting media.

3. It would appear that the active ingredient was a thermo lable substance found in the seropositive sera, and inhibited, but not completely destroyed by heat at 56° C for 30 minutes.

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Editorials

Our New President For 1947-1948

Preston Thomas Brown was born in Denver, Colorado, in 1905. He received his medical degree from the University of Colorado School of Medicine in 1928, and took his interneships at the University Hospital of the State University of Iowa and at Memorial Hospital, New York City. He returned to the University Hospital at Iowa City for a residency in obstetrics and gynecology, which was completed in 1933, following which he located in Phoenix, where he practiced these specialties until his enlistment in the Army in 1942.

He entered the Army with rank of Major and after the usual training service in the United States, he went overseas with the 108th Station Hospital unit as chief of the surgical service. Later he was commanding officer with this unit as chief of the surgical service. Later he was commanding officer with this unit in Australia, New Guinea, Philippines and Japan. At termination of the war, he was honorably discharged with rank of Lieutenant-Colonel.

Dr. Brown is a diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons. He has served and is still serving in places of responsibility in Maricopa County medical circles. He is an active member of the staffs of all three general hospitals in Phoenix and at present is chairman of the general staff at St. Joseph's Hospital. Upon resumption of practice in Phoenix he is confining his work to medical and surgical gynecology. A capable administrator in any office he has filled in civilian practice and in army service, he will ably discharge the important duties of the highest office in medical organization in Arizona.

"Voluntary Medical Prepayment Plans as They Apply to Rural Communities"

by

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A review of the history of voluntary medical prepayment plans reveals that in their modern concept they are strictly American. The oldest of American plans is the Northern Pacific Railroad Benefit Association which was organized in 1882 and has operated continuously since that date. Prepayment plans have developed through industrial establishments, universities, consumer and cooperative groups, various governmental units, Blue Cross and medical societies or groups of physicians. In addition, insurance companies have offered various types of health and accident coverage to the public. The medical

This is a copy of a talk given in Chicago Friday morning, February 7, 1947 at the Second Annual National Conference on Rural Health, sponsored by the Committee on Rural Medical Service of the American Medical Association.

profession has contributed to the successful operation of all of these plans.

Efforts are being made to obtain accurate figures on the number of people covered by these various voluntary prepayment plans. At present any figures must be considered as estimates as no way has yet been devised to eliminate duplications in cases where people have subscribed to two or more of these plans.

I am indebted to Mr. L. S. Kleinschmidt, of the Division of Prepayment Plans of the Council on Medical Service, for the factual data given in this paper. The closest estimates obtainable today indicate that the total number of persons covered by he various types of voluntary prepayment plans is as follows:

Type of Plan	Total Coverage	Year of Information
Industrial	1,516,000	1945
University Student	100,000	1943
Consumer and Cooperative	504,000	1943-45
Governmental	580,000	1945-46
Blue Cross	25,000,000	1946
Medical Society & Physicians Groups	5,000,000	
	32,700,000	
Insurance companies, hospital, surgical, accident and health coverage	44,000,000	
Total	76,700,000	

Progress was slow in the beginning. Expansion of plans as we know them today has occurred largely during the past ten years and the major increase in enrollment in the past three years.

The prepayment plans for medical care received their first impetus as a result of the phenomenal success of the Blue Cross Hospital Service Plans. With both the hospital and the patients in need of some sort of program to ease the burden of hospitalization costs, these plans grew rapidly. Today the term "Blue Cross" is recognized by almost everyone in the United States.

Using the experience of prepayment hospital plans as a guide, a number of state medical societies undertook to develop medical care plans. The first statewide plan was the California Physician's Service organized in February, 1939. Within a year Michigan Medical Service was organized. These two plans, together with the county medical society bureaus of the States of Washington and Oregon gave the medical profession the necessary experience upon which to build what has now become a nationwide movement.

The growth of medical care prepayment plans since 1939 has almost paralleled that of the hospital service plans. It took Blue Cross between five and six years to place thirty-eight plans in operation. This compares favorably with the six years it took to develop thirty-seven medical care plans. The same comparison can be made with reference to enrollment. In their first seven years medical care plans enrolled 2,845,000 while Blue Cross enrolled 2,870,000.

The development of prepayment medical care was in many ways more difficult and complicated than that of hospitalization. Hospital care is a more unified service with relatively few procedures involved, whereas medical care included an almost unlimited variety of services. The problems involved in the payment and handling of claims for medical procedures are far more difficult than those for hospitalization. Years of experimenting with various methods has resulted in definite progress toward essentially sound advancement. Hospital care plans deal with a relatively few institutions whereas medical care plans deal with several hundred or even thousands of doctors. A medical care plan then to be practical must not rely upon a relatively uniform institutional point of view but actually upon a host of individual attitudes if we are to retain a high quality of medical care.

The growth of voluntary health care insurance plans since 1939 has been most remarkable. Counting the 28 local plans in Washington and Oregon as two statewide plans, there are now 58 medical care plans sponsored by medical societies or *Blue Cross*. Plans are now in operation in 33 states and are in the process of formation in 13 more states and the District of Columbia. This leaves but two states, Mississippi and South Carolina, without some development reported sine 1939 and that the rate of growth has been rapid and steadily increasing attest to the application of common sense and to the desire of the medical profession to have prepayment medical care of a high quality available to all the people.

The A.M.A. Ten Point Program

The House of Delegates of the American Medical Association in their annual session at Chicago in 1945 adopted the following resolution: "The Board of Trustees and the Council on Medical Service will proceed as promptly as possible with the development of a specific national health program with emphasis on the nationwide organization of locally administered prepayment medical care plans sponsored by medical societies."

The Ten Point Program recommended: 1. Minimum standards of nutrition, housing, clothing, and recreation are fundamental to good health. 2. Preventative medical services should be available to all and should be rendered through professionally competent health departments. 3. Adequate prenatal and maternity care should be made available to all mothers. 4. Every child should have proper attention including scientific nutrition, immunization, and other services included in infant welfare. 5. Health and Diagnostic Centers and hospitals necessary to community needs are preferably supplied by local agencies. 6. Voluntary health insurance for hospitalization and medical care is approved. 7. Medical care, including hospitlization to all veterans should be provided preferably by a physician of the veteran's choice. 8. Research for the advancement of medical science including a national science foundation is endorsed. 9. Services rendered by volunteer philanthropic health agencies should be encouraged. 10. Widespread education in the field of health and the widest possible dissemination of information regarding the prevention of disease and its treatment.

In order to implement the sixth point relating to voluntary health insurance, the Council on Medical Service established certain minimum standards of acceptance. These are: approval by the state or local medical society in the area in which it operates, the assumption of the local medical profession of the responsibility for a high grade of medical care, the free choice of physicians, the honest use of promotional material, the retention of the sanctity of the physician-patient relationship, accepted enrollment practices, and clarity of benefits. Plans meeting these requirements will be soundly organized. The plans which meet and continue to meet these requirements are given the "Seal of Acceptance of the Council on Medical Service of the American Medical Association. Provisions are made for the frequent review of the plans and any found violating these fundamental principles of sound procedure may have their seal of acceptance revoked. On the other hand the Council, being more concerned with healthy expansion of plans and benefits provides information and aid to all accepted plans and others seeking acceptance so that their service to the health needs of their communities may be continually broadened.

Methods of Expansion.

With the experimental phases of medical care plans well under way more attention can now be devoted to expansion of enrollment and expansion of benefits with special attention to reaching rural subscribers. The Council on Medical Service has established a Division of Voluntary Prepayment Medical Care Plans which acts as a clearing house of factual information, experience, and successful practices of all operating plans and, by the constant interchange of this information, enables the rapid correction of unsatisfactory practices.

The expansion of enrollment and the expansion of benefits have been termed by Dr. H. H. Shoulders, President of the American Medical Association, as the vertical, which involves the question of how high up the scale of benefits indemnities or services can go under the insurance principle and how high the costs can go to cover these increased services; and the horizontal, involving the question of how far it is necessary to go to reach all potential subscrib-

The early plans in Washington and Oregon included practically all medical services, surgery, home and office care, and even limited nursing and dental care. Throughout the years this broad coverage has been continued. The plans organized in 1939 and 1940 followed this lead and also provided for reasonably complete medical care. In contrast to the experience in the Northwestern states, the experience of the latterly formed plans with full coverage was unsatisfactory. It was found that the public would not pay the premiums necessary to carry out such a broad contract. Whenever a surgical contract plus a general medical care contract was offered to the public, the ratio of enrollment was more than 100 to 1 in favor of the surgical program. As a result the plans have generally preferred to begin operations with fairly restricted coverage such as surgical care, obstetrical, x-ray, and anesthesia in hospitalized cases. Now most of the services necessary in cases so severe as to require hospitalization are covered.

The tendency to limit services to those performed in the hospital is a natural one. In the first place most of the costly illnesses are those that require hospitalization. Secondly, there is going to be little abuse of such services. Third-

ly, the actuarial experience in hospitalized cases is sufficient to provide plans with a more certain basis for determining adequate premiums. A recent study of fifty-one medical service plans shows that thirty-four of the plans offer surgical services, obstetrical and specified medical services in the hospital. This same study shows that plans are continuing to expand benefits. Twelve of the plans provide for general medical care, such as home and office calls, and five plans provide almost complete coverage.

The average premium for a single subscriber is approximately $1.25 and ranges from as little as sixty cents to $4.85 a month. Family coverage ranges from $1.35 to $10.00 a month with an average of about $3.00.

Plans are still experimenting with benefits and with premium rates. As one plan succeeds in some new idea, the basis for this success is made available to other plans. The same, of course, is true of failures. In this manner vertical expansion will continue to grow as actual experiences and public demand dictate.

Reviews of experience in rural areas have indicated that with the facilities available the rate of use under prepayment is higher than in the cities. The accumulation of physical defects and of chronic illness is greater. The risk of accidents is greater. Farmers are not usually covered by Workmen's Compensation. All injuries and illnesses automatically come within the scope of the prepayment plan.

The success of a plan offering general medical coverage depends on meeting the people's need for medical care with proper control of abuses and on the maintenance of the costs at a point consistent with adequate standards of service and at premiums salable to rural subscribers. It is the job of the medical profession to keep its members informed. So too, it is the job of the enrolling groups, whether they be factory, farm, or community to prevent the abuse of the benefits of the plans by their members. Equal interest in determining both coverage and premiums on a local basis should facilitate answers as to what additional medical services are needed, what the subscribers will pay, and what the plan charge must be to remain financially sound.

Horizontally, the expansion of the prepayment medical care program is limited only by the ability and willingness of the public to enroll as subscribers. It is true that most of the plans have tended to concentrate on urban areas where large group enrollment was possible. This was a sound method because the sooner a plan obtains an adequate risk spread, the sooner it can expand its benefits and enrollment program. Large group enrollment provides this spread more easily and with less acquisition cost.

However, the pattern is now changing and having reached a reasonable enrollment and a sound financial level, ways and means of reaching all population groups—particularly the rural—are being explored.

In general, the pattern has not been to deal with the farm group as a separate problem but to include them in what is called community enrollment. In community enrollment the farmer is considered a part in the area encircling a town or village. Usually the town is the center for trading. The whole community then becomes a group with premium payments payable at some central place.

A Method for Proceeding

A successful Blue Cross enrollment campaign in rural areas was carried out in Colorado by the Weld County Agricultural Health Association. Enrollment was on a community basis reaching all elements of the community as a civic service. Weld County, the largest county in Colorado, is about three times the size of Rhode Island and has a population of 63,700. Greeley is the largest town in the county with 15,900 population, and there are several smaller towns. Prominent businessmen, farm organization leaders and various club leaders met with the Blue Cross representatives and decided to form a Health Association under the Colorado law governing cooperatives. Twelve directors, each representing a definite district in Weld County, govern the Health Association. This division provided workable units in size and population for an enrollment and administrative program. Enrollment was not limited to Blue Cross but each district was allowed to enroll separately in the Colorado Medical Service plan whenever fifty per cent or more of the families in the district had subscribed to the Health Association. To date Weld County has 8,000 participants enrolled. Colorado has organized ten other similar county health associations and thus have enrolled one in every six persons in

rural areas and one out of every two urban residents.

Iowa has organized fifty-five similar County Health Improvement Associations. Other states are enrolling on a similar community basis with less formal organization.

Voluntary prepayment plans are growing rapidly in rural areas. A survey of Blue Cross Rural enrollment reported in November, 1946, by the Blue Cross Commission shows an increase of 200 per cent since 1944. The study shows a total of 1,637,533 subscribers and dependents now as compared with 500,000 rural members in 1944.

Many medical care plans have developed their organizations to the point where rural enrollment is being carried on or plans are being made by those reaching into rural areas. A study of these plans now under way indicates that about one-half of those doing rural enrollment, twelve plans, have reported 304,542 subscribers and dependents. It is estimated that the study, when completed, will show medical care plans in a position comparable with Blue Cross of two years ago—or a half million rural members.

The problem cannot be solved at a national level. It must, to be successful, be worked out in the community where the need exists. There must be developd a community responsibility which will lend its active support if any effort is to succeed.

Continuing improvements in transportation and extension of better roads will accelerate the growing tendency of rural people to bring the patient to the physician. Thus the physician's care and the use of other health facilities can be extended over an area that includes a sufficient number of people to meet the costs of the services provided.

Use of the Insurance Principle

A study of the fees for medical services rendered by the rural practitioners shows they are not excessive. However, there are times when illnesses strike and the severity of the cases cannot be predetermined. For this reason many farm families faced with severe illness find it difficult to meet the full costs from their income. This problem of meeting the costs is basically the same as it is with people living in towns. The only sound method so far devised is through the use of the insurance principle we have described. Prepayment medical care plans available to all people of a community, towns people and farmers alike, tend to spread the risk and distribute the costs. Thus, having the cost known in advance, proper planning and budgeting for these otherwise unpredictable costs is possible.

Families or individuals unable to pay the full costs of medical care from their incomes and those now receiving public assistance should be called upon to pay for their own medical care as far as is consistent with their incomes and provision made through locally administered public funds to pay the balance of the premium required to participate in a prepayment plan. Every effort should be made to help and not to pauperize or regiment the most individualistic element of our social economy—the American Farmer.

The problem of the lower income groups can be solved by local communities and state actions in line with the rapidly developing voluntary prepayment medical care movement.

Division of Responsibility

Who carries the responsibility for the medical care program? The physicians because of their special training cannot escape the responsibility for the professional aspects of the program. Experience has shown that they do not want to. The recipients of the medical service (the people) cannot escape the responsibility of paying the costs of the services. Both should do their full share in providing the needed facilities. With full cooperation of the medical profession and the people, working in mutual confidence, a medical care program can succeed by undertaking the various types of medical service one step at a time. As experiene is gained, additional types of medical care can be included. Coverage may be as complete as is desirable in any community. Progress can be as rapid as experience indicates and the economic situation permits.

For ten years or more doctors have operated experimental plans in medical care insurance. On the basis of this experience they now feel ready to recommend prepayment plans to the people. The Council on Medical Service created by the House of Delegates of the American Medical Association has as one of its primary functions the fostering and developing of prepayment plans. The American Medical Associations have rural health committees devoting their attention to the rural problem as do the various State Medical Associations.

It Can Be Done

Local representatives of farm organizations such as those present here today, together with business groups and local medical societies working in full cooperation and mutual confidence and understanding are now in a position as never before to work out this heretofore difficult phase of delivering medical care to all the people. And unfettered by the totalitarian idealogies of the old world we will continue to be "America, the land of individual opportunity."

Industrial Health Meetings

A conclave of combined professional personnel in industrial health work over the entire nation will take place at the Hotel Statler, Buffalo, N. Y., April 26 through May 4, 1947.

These meetings will represent the 32nd annual gathering of the AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS; the 9th annual conference of the AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS; the 8th annual meeting of the AMERICAN INDUSTRIAL HYGIENE ASSOCIATION; the 5th annual conference of the AMERICAN ASSOCIATION OF INDUSTRIAL NURSES, and the 4th annual meeting of the AMERICAN ASSOCIATION OF INDUSTRIAL DENTISTS.

The sessions will be replete with many new subjects of interest, including among others, round table discussions for chemists, engineers, physicians and nurses; a symposium on new problems in the developments of industrial hygiene; a discussion of state codes and industrial hygiene administration; conferences on environmental control, on particle size, and analytical procedures; clinics on fractures and traumatic surgery, including a symposium on back problems; hazards incident to the use of the atomic bomb; reports on the Bikini experiments with motion pictures; tracer chemistry in toxicological research and experience with range finding tests; progress in the teaching of industrial medicine in American medical schools; the development and administration of industrial dental clinics in various industrial groups; a panel discussion on new preventive measures in industry; a penal discussion on in-service education of the nurse in industry, and many other subjects which can be found by consulting the preliminary program.

Prominent speakers on important subjects will be featured at dinner sessions, including other events such as the Cummings Memorial Lecture and the presentation of the Knudsen Award for the Most Outstanding Contribution to Industrial Medicine during the past year.

Also available at this meeting will be the opportunity to inspect and study a splendid group of scientific and technical exhibits, with the most recent developments and medical department accessories.

Further details and a copy of the preliminary program may be secured by writing to Dr. Edward C. Holmblad, Managing Director of the American Association of Industrial Physicians and Surgeons, 28 East Jackson Blvd., Chicago 4, Ill.

All hotel reservations are made by the Housing Bureau, Buffalo Convention and Tourist Bureau, Inc., 602 Genesee Bldg., Buffalo, N. Y.

(Prepared for announcement of coming meetings in Buffalo, N. Y., April 27-May 4, 1947.

Office of the Surgeon General

ARMY REPORTS ON USE OF STREPTOMYCIN

The new anti-infection agent, streytomycin, which is in the same general class as penicillin, appears effective in appropriate doses against more than half the infective bacterial organisms ordinarily encountered by surgeons, according to the report to The Surgeon General's Office from the Halloran General Hospital. Clinical studies of the use of the drug throughout the army have been submitted and evaluated at Halloran.

On the other hand, it apparently has specific poisonous effects when given over an extended period, and bacteria soon become resistant to it so that it probably can be used only with maximum effect within a limited period on the same patient.

The drug became available only late in the war and is still scarce and expensive.

Army experience with the drug is probably the most extensive to date due to the ability to compile and follow results. Outside the services because of limited opportunities to observe results in large numbers there have been varied, and sometimes quite exaggerated, reports on its value and it often is referred to popularly as a "miracle drug." From the first careful observations of its effects have been carried out by army doctors by orders of The Surgeon General, and these are being continued. The findings to date

are summarized in the Army Medical Bulletin of November 1946.

The observation of the ability of bacteria to develop resistance to the drug after a few days may be of particular importance at this time. The same has been noted in respect to both the sulfa drugs and penicillin, but apparently the phenomenon is more pronounced with streptomycin. In at least one case, test tube experiments showed, there was a 100-fold increase of the resistance value for a particular type of infection in an individual for the rest of his life. Improper use may cause variation and selection in disease agents so that streptomycin is no longer effective for the infection where it is of greatest value at the present time.

Bacteria, on the basis of certain chemical reactions, ordinarily are divided into two classes—gram positive and gram negative. The new drug, in test tube experiments, seems effective in varying concentrations, against 60 per cent gram positive and 80 per cent gram negative organisms ordinarily encountered in surgery.

Of paramount importance, is determination whether a specific micro-organism is susceptible to the drug before it is administered by mouth, by injection, or direct application.

The army experience bears out previous claims that streptomycin is of especial value in clearing up infections of the urinary tract, provided that the organisms causing the infections are susceptible ones. If the treatment is not entirely effective in three days ordinarily no good results can be expected from its continuation. In gonorrheal infection which has proved resistant to both sulfadiazine and penicillin outstanding results have been obtained.

Use in army hospitals gives no support to claims that the drug is of value in infections of the prostate. The drug is not concentrated in that organ.

It was found to have very little value against bone infections, except when used in conjunction with surgery where there could be direct application.

Thus far streptomycin has not given dramatic results in peritonitis, but its continued use as an auxiliary treatment seems justified.

In various dysenteries due to susceptible bacteria considerable benefit has been noted, sometimes when the drug is given by mouth alone.

In septicemia—still provided that the organism responsible for the infection is a susceptible one—streptomycin has proved very effective, but it is still essential that unapproachable foci of infection be removed by surgery.

The substance has little value, so far as the army experience goes, against typhoid fever and it is apparently of no use in controlling carriers of this disease.

In undulant fever there have been apparent clinical arrests of the infection from the combined use of streptomycin and sulfadiazine after each drug given alone had failed. Further study will be required, however, before any valid conclusions can be reported.

It is very effective against tuleremia, or rabbit fever, provided the specific organism responsible has been demonstrated in test tube experiments to be susceptible to the drug.

Up to date experience with only a few cases of meningitis have been reported and the results, in conjunction with other treatments, have been quite good. The Army doctors found, however, that it must be given by injection into the space between the thick membranes surrounding the brain and spinal cord and the brain or spinal cord tissue. Circulating in the blood stream, it cannot pass this barrier to reach the infecting organisms.

Excellent results have been obtained with direct application of the drug to infections of the external ear, the pleural cavities and the brain. Infections elsewhere will not reach local foci of infection in sufficient concentration to be effective.

One of the hopes of the medical profession has been that streptomycin would prove of some value against tuberculosis. The army experience neither confirms nor refutes this since a much longer series of investigations will be required before there can be any valid conclusions.

Balances against the demonstrated value of streptomycin in suitable cases are some apparently toxic effects. Some of these are probably due to impurities in the drug but others seem to be specific for the drug itself. The most serious of these is what seems to be an irreversible damage to part of the eighth cranial nerve which appears when streptomycin is given in large doses by injection for more than ten days. This means that one's sense of balance may be disturbed for a long time, with possible attacks of dizziness and nausea. This was found in two army cases. A third patient showed partial deafness, indicating that there had been a poisonous

effect on the other portion of the eighth cranial nerve, which is the path of hearing. Toxic effects also were noted on the kidneys. All this demonstrated that the drug should be given only by physicians, and then only after careful consideration of the organisms involved and the safe dosage.

NEW WATER-PURIFYING TABLET DEVELOPED BY ARMY

A new tablet for purifying water in the soldier's canteen which is considered superior to the chlorine-type tablets used during the war, has been announced by the War Department.

With iodine employed instead of chlorine, the new tablets make the drinking water less objectionable in taste and odor. Tests have shown that the iodine-containing tablet has greater sterilizing flexibility in that it can be used under a wide range of conditions. It is also more suitable and dissolives more quickly than its predecessor. This tablet was developed by scientists of the Army Medical Department, Quartermaster Corps and Corps of Engineers.

When the chlorine-containing tablets were dissolved in water, soldiers complained that they made the water unpleasant to the taste. It was found that even with strict supervision it was sometimes difficult to prohibit the soldiers from drinking water from streams or wells of questioned purity.

After examining a large number of different compounds, scientists found that triglycine hydroperiodide possessed the highest military characteristics. Quickly dissolving tablets containing this chemical which liberated seven and one-half parts per million of elemental iodine were subjected to extensive testing. It was found to be a safe and highly effective agent.

Additional tests of the new "tablet, water purification, individual, iodine," as the compound has been designated, will be carried out during the ensuing year.

SURGEON CATALOGUES NERVOUS REACTIONS

Seven distinct nervous reactions developed among the men who flew "the hump"—the air supply route from India to China across the Himalayas—during the war.

Continuous service on this cloud road probably provided the most severe nerve strain endured by Army air men and study of the observed symptoms, as reported to the Surgeon General's Office by Major William M. Jeffries of the Medical Corps, a Flight Surgeon of the Air Transport Command in the China, Burma, India Theater, discloses what might be expected when men are pressed toward the limits of human endurance.

The fact that many showed no symptoms at all, Major Jeffries stresses, is a high tribute to the men who composed this force.

The "hump" route, as he describes it, was over a series of high, jagged mountain ranges divided by rivers flowing through precipitous gorges. There were no emergency landing fields. Much of the way was over Jap-held territory. Storms frequently blew ships far off course. Icing conditions were often bad, yet de-icing equipment had been removed from most planes. Flights were made at altitudes of from 17,000 to 35,000 feet, yet oxygen equipment was often inadequate; usually passengers had none. Landing fields were endangered by low ceilings and ground fog. Surrounding high mountains allowed no margin for error on instrumental letdowns. On the ground, heat and humidity were so great that clothing was constantly drenched with perspiration. Prickly heat was almost universal and sleep almost impossible. There was constant danger from snakes and insects—certainly annoyances.

The Combination of these factors, says Major Jeffries, was sufficient to shake the stability of some of even the soundest individuals, a large number of psychological disorders developed, and it was remarkable that more were not encountered.

Psychiatric cases were occurring in large numbers in Italy in the late winter and spring of 1944. This led to a clinical study of individual cases, and a study of men exposed to the same stresses who showed no abnormal nervous reaction.

There is no such thing, the psychiatrists conclude, as "getting used to combat." In the North African Theater, for example, practically all infantrymen who were not otherwise disabled ultimately became nervous casualties. There was general agreement between both medical and line officers that 200 days in combat, not necessarily continuously, represented about the limit of human endurance. By that time if a man had not definitely "cracked up" he had become so jittery that he was ineffective as a

soldier and a demoralizing influence among his comrades.

The British, however, believed that a man could stand 400 days and attributed the difference to their policy of pulling an infantryman out of action at the end of 12 days or less for a rest of at least four days. American infantrymen in Italy frequently stayed in combat continuously 30 to 40 days and occasionally as long as 80 days.

The report recommends that the limit of combat for any infantryman be 210 days—and this only in extreme emergency. It also urges that special recognition and privileges be given infantry soldiers, that greater efforts be made in indoctrinating soldiers and the categories and channels of evacuation be clarified for both line officers and Medical officers to the end that true malingerers will not be evacuated and severe psychoneurotics not retained in their combat units.

The war ended before recommendations of this report could be put into practice.

MANY AMERICANS IMMUNE TO MUMPS

Thirty per cent of the American people probably have had mumps without knowing it. The result has been a high degree of immunity to epidemics of this common, but sometimes quite serious, disease of childhood. Such is the conclusion from studies of 50 groups of children and adults conducted by University of Pennsylvania and Harvard University medical scientists under a contract with the Commission on Measles and Mumps of the Surgeon General's Office, United States Army. Mumps and measles usually are paired as childhood maladies. Each is caused by a specific filterable virus. Both diseases are very contagious. One virus presumably is as widely disseminated in the population as the other. Yet the studies reported by the Doctors Elizabeth P. Maris, John F Enders, Joseph Stokes, Jr., and Lewis W. Kane, show that about 33 per cent of young adults have a probable acquired immunity to the disease indicating some past infection of which they were unaware. One attack of mumps is believed to protect an individual against further attacks of the virus for the rest of his life. Statistical studies have shown that whereas about 90 per cent of the American population suffer from measles at some time or other, only 60 per cent are victims of mumps. The immunity of a person was determined by the so-called "complement-fixation" test of the blood serum with mumps virus cultivated in incubated chicken eggs, and also by a skin test with similar material. In this hidden reservoir of acquired immunity mumps seems to bear some likeness to poliomyelitis, also a virus disease of children. It is believed that about 90 per cent of the population have had polio in a sub-clinical form at some time, with the result that they are permanently protected against it. The reasons why mumps should attack some persons in such a mild form that it is not recognized—it may amount to no more than a slight headache or an "out-of-sorts" feeling—is unknown. The technique of determining immunity may prove of considerable value in times of mumps epidemics when the relative susceptibility of a population can be determined before undertaking defense measures.

AMERICAN CONGRESS ON OBSTETRICS AND GYNECOLOGY

The program of the Third American Congress on Obstetrics and Gynecology to be held September 8-12, 1947, in St. Louis will feature general sessions for all groups making up the congress as well as smaller individual group meetings and round table discussions. The morning sessions will be panel-type presentations of the following subjects: Tuesday, Sept. 9: Anesthesia and Analgesia; Wednesday, Sept. 10: Cancer; and Thursday, Sept. 11: Caesarean Section.

The afternoon meetings of the medical section of the Congress will consider on Tuesday: Psychosomatic Aspects of Pregnancy; on Wednesday: Pregnancy complicating Cardiac Disease, Diabetes and Tuberculosis; and on Thursday: Recent Advances in Endocrinology.

Round Table discussions from four o'clock to five daily will consider such topics as etiology of abortion, asphyxia, fibroids, prolonged labor, infertility, early ambulation, adolescence, treatment of abortion, genital relaxation, ovulation, the menopause, the cystic ovary, uterine bleeding, nutrition in pregnancy, geriatric gynecology, endymetriosis and erythroblastosis.

Concurrent sessions and round tables for nurses, hospital administrators and public health workers are being arranged.

The popular forceps and breech demonstrations that attracted so much attention at the Second Congress in 1942 will be increased in number so that eighteen demonstrations per day will be held, six each at nine, one and five o'clock daily.

A large Scientific and Educational Exhibit is being set up under the direction of Dr. J. P. Pratt of Detroit and a comprehensive Motion Picture Program is being arranged by Dr. John

Parke of Washington, D. C. The committees assisting these doctors will review applications by prospective participants late this spring. Anyone wishing to make application for space in the Scientific Exhibit or for time on the Motion Picture Program may obtain the proper blanks from the office of the Congress at 24 West Ohio Street, Chicago 10, Illinois.

NOTICE

The U. S. Food and Drug Administration wishes to bring the following notice to the attention of physicians. Analbis Suppositories are involved in the deaths of about 14 children under six years of age. The trouble may be due to excessive doses of the drug. However, pending full investigation, the manufacturer, Specific Pharmaceuticals, Inc., 331 Fourth Ave., New York City, is recalling all outstanding stocks, and the Food and Drug Administration is assisting them. There are still quantities of the drug in possession of pharmacies, hospitals and physicians. Any physician who does have this drug is urgently requested to return it to the manufacturer for credit.

ANNUAL MEETING, AMERICAN COLLEGE OF CHEST PHYSICIANS

The Thirteenth Annual Meeting of the American College of Chest Physicians is scheduled to be held at the Ambassador Hotel, Atlantic City, New Jersey, June 5 to 8. An interesting scientific program has been planned for this meeting. Prominent speakers from other countries will present papers.

The oral and written examinations for Fellowship will be held on the first day of the meeting, June 5. Applicants for Fellowship in the College who plan to take these examinations should communicate *at once* with the Executive Secretary, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

The Convocation for new Fellows and Life Members of the College will be held on Sunday, June 8. At this time certificates will be awarded to Fellows and Life Members admitted since June, 1946.

AMERICAN ACADEMY OF ALLERGY

The American Academy of Allergy, in cooperation with the Medical Faculty of the University of California, will offer an Orientation course in Clinical Allergy for General Practitioners at the University of California Hospital, San Francisco, from July 7 to 11 inclusive, 1947, under the auspices of Medical Extension, University of California. This course (which) has received the approval of the Committee on Education of the American Academy of Allergy (will be announced in detail in a later issue of this publication.)*

STACY R. METTIER, M.D.
Head of Postgraduate Instruction
Medeal Extension, University of Cal-
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* As you wish, and feasible

ABSTRACTS

Journal, American Medical Association.
131:1419-1420 August 24, 1946

Renal Damage Due to Ischemic Muscle Necrosis.

MAJOR WM. MOLONEY, MAJOR SIDNEY L. STOVALL and MAJOR DAVID H. SPRANG, JR.

Stimulated by the work of Bywaters and other British investigators, the present authors present case representing the so-called "crush syndrome" or ischemic muscle necrosis syndrome, which occurred in a soldier aged 23 years, who suffered a severe compound fracture of the right femur and extensive soft tissue damage. The femoral artery was found to be in severe spasm in Hunter's canal, during the operation for debridement, some hours after his accident, which was associated with thrombosis of the femoral vein. A Leriche periarterial stripping was done, which resulted in a return of pulsation to 1" above the distal end of Hunter's canal. Intensive parenteral treatment with plasma, blood, antibiotics and chemotherapy was instituted and continued throughout the course of his critical period. Urine passed 2 days later was dark orange in color and contained large numbers of pigment, containing casts. A benzidrine test gave a strongly positive reaction. Some edema of the opposite ankle appeared, which was followed by a tense firm swelling over the posterior left leg on the following day. Incision for suspected hematoma revealed only that the muscles were under great tension. In the meantime a guillotine amputation of the affected extremity (right) was performed.

The urinary findings (above) suggested the true nature of his condition and subsequent lab-

oratory investigations of the patient's urinary specimens at Cambridge University confirmed the presence of myoglobin (and not oxyhemoglobin) on spectroscopic studies.

During an apparently satisfactory convalescence the patient suddenly developed, on his 9th day, painful cyanosis of the left foot which gradually demarcated and necessitated amputation of the distal 1/3 of the foot; following which he made a slow recovery.

The authors point out the fatal tendencies in renal damage following crush injuries and the urgent necessity of its prompt recognition in order that adequate treatment may be instituted. They explained the vascular spasm on the apparently uninjured side as a reflex mechanism. The nature of the toxic damage to the kidneys is not clearly understood. Alkaline diuresis (as was established in this case) is the first line of defense against such renal damage, according to Bywaters, and perhaps saved this patient's life. This condition must always be suspected and proper urinary studies be carried out, along with other studies in an attempt to throw further light upon this interesting and often serious condition.

J. Kulowski, M. D.

JOURNAL OF BONE AND JOINT SURGERY

Vol. 28, No. 3; Old Series, Vol. 44 Page 550
July, 1946

Knee Fusion by the Use of a Tree-Flanged Nail

David M. Bosworth

Orthopedic Service of St. Luke's Hospital
New York

The author presents a method of arthrodesis of the knee in which a three flanged nail of the Smith-Petersen type is used to secure stability and continuous apposition, thereby hastening the arthrodesis. This type of operation has been done in eighteen patients by the author. Fourteen of these cases are presented. In these fourteen cases good fusion was secured in all but one, and this was a case of Charcot joint with severe disintegration.

The operative procedure is carried out through a horse-shoe incision. The articular surfaces are removed and the joint debrided if necessary. A Smith-Petersen nail is inserted in an oblique medio-lateral direction through the joint line. The posterior two-thirds of the patella is removed and may be used as a free graft. The posterior surface of the remaining one-third of the patella with ligament attached is placed across the joint line in contact with the previously denuded upper end of the tibia and lower end of the femur. A plaster boot is applied, the foot portion being removed in ten days to three weeks, so that only a cylinder cast is used for the succeeding three months and the patient may be ambulatory with crutches.

This type of arthrodesis is recommended for Charcot joints, infections, arthritis, osteoarthritis, residual poliomyelitis and the relaxed knee of hydrocephalus. It is not recommended for tuberculosis of the knee because of the danger of concurrent secondary infection. It is likewise not to be used where an open epiphyseal line has to be crossed or in cases where osteomyelitis has been present or a suppurative arthritis is still active. The great advantage of maintenance of continuous apposition of the surface without a bone graft, the fact that only a simple plaster boot is required and the consequent shortening of hospialization all combine to recommend this as an attractive procedure.

S. L. STOVALL, M. D.

SURGERY, GYNECOLOGY AND OBSTETRICS

August, 1946

The author studied changes which take place when preserved ox fascia is placed in contact with living human tissue. In this case implantations were done during various operative procedures, especially for hernia as advocated by Harrison. Two x 2½" pieces of ox fascia were aseptically prepared and preserved in 70% ethyl alcohol for two weeks; immediately after the animal was killed. The implants were removed at subsequent reoperations at intervals up to four years in duration. The author found that the preserved fascia rapidly becomes adherent, and is gradually replaced by the ingrowth of fibroblasts and capillaries and the formation of human connective tissue. But, this process is still incomplete after four years, at which time the original fascia can still be identified. During this process of incorporation there is surprisingly little foreign body reaction. The value of preserved fascia, however, as a framework into which normal tissue may grow, in various plastic procedures, is pointed out concretely in these observations.

The author recalls that the use of dead material as grafts has been based on the work of Nageatte and Sencert. The former argued (1916) that all connective tissue substances were fundamentally non-living in character and origin. He therefore believed that, since connective tissue substances are inert coagula formed from living cells (like blood plasma) that both fresh and preserved connective tissue grafts would become incorporated so perfectly, by a process he (Nageatte) called "reviviscence", that they could

noa later be distinguished from the surrounding recipient tissues. The experimental work of several others, in support of this contention, is mentioned. The observations of the present author do not, however, agree in this nor did he find anything to back Nageatte's theory of the nature and origin of the connective tissue, although the value of the preserved tissue was quite evident. The significance of alcoholic preservation was learned from animal experiments of Kooutz, who believed that the alcohol eliminates the antagonistic action of foreign protein, since he did not note any difference between heterogenous and homogenous grafts under the conditions of his experiments.

J. KULOWSKI, M. D.

1. "The Fate of Preserved Heterogenous Grafts of Fascia when Transplanted Into Living Human Tissues." Jacob Chandy, M.D., B.S., M.Sc.: Philadelphia, Pa.

ORGANIZATION SECTION

CHARLLS P. AUSTIN, M. D., President

SOCIETY ACTIVITIES
MARICOPA COUNTY MEDICAL SOCIETY
1947

President ..... Dr. O. W. Thoeny
Vice-President ..... Dr. Henry G. Williams
Secretary-Treasurer ..... Dr. L. L. Tuveson

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Dr. James Lytton-Smith '47 — Dr. James R. Moore '49
Dr. Louis Jekel '48

ADDRESS BY DR. JAMES R. MOORE
Retiring President of Maricopa County Medical Society

After having served as your President for the past year, it seems fitting that I should review some of the activities of the Society during my incumbency and make such observations and recommendations as appeal to me to be proper and for the good of the Society.

During the past year, our Society has grown greatly in membership and it has been gratifying to note the excellent attendance, including those recently returned from service, as well as the many newcomers who, since the war, have chosen Phoenix as their home. Not only has the Society grown in numbers, but with this growth, there has been an increase in the volume of business and number of problems presented. This has reached such proportions that it has been difficult to crowd into one monthly meeting, all the business of the Society and have any time left for a scientific program. To relieve this situation, I believe it would be advisable for the Board of Directors to hold regular stated monthly meetings, preferably about the middle of the month, at which time all business and all proposals should be presented. The Board of Directors should be empowered by the Society to act directly upon all minor matters. The Board of Directors should present to the Society at the regular meetings, all matters of major importance and all questions of general policy, along with their carefully considered recommendations. In this manner, more prompt action could be secured on routine matters and more time would be allowed for the discussion and deliberation upon important subjects by the Society as a whole.

Frequently, the President or the Secretary are approached by members of the Society regarding the propriety and ethics of certain contemplated acts or procedures, the answers to which often require great discrimination, judgment and familiarity with technical details. The President or the Secretary may or may not possess these qualifications but, even so, the approval or disapproval of any proposed act throws too great a responsibility on one individual. I believe it would be well to have appointed a committee of three members, who are experienced and well versed on these matters, to whom such inquiries could be directed. The duties of this committee might well be expanded to include a few lectures to the Society in general and to the probationary members in particular, if such a status be established by proposed changes in our By-Laws, on the general subject of medical ethics, melical liability and jurisprudence.

During the past year, plans were inaugurated for the continued expansion of the library, particularly for the establishment of a permanent home for the library and the Society. While it is not contemplated that this home can be established in the immediate future, we should never be-

less continue to actively plan for its development and be ready to initiate the program whenever a favorable occasion arises.

To our new members and to those recently returned, attention is called to the group insurance plan covering health and accident, available to them under liberal terms. This insurance has been operative during the past year with satisfactory service to participants; and those not already enrolled, are urged to obtain full details regarding this service from the Secretary.

The Salt River Valley Blood Bank has completed its third successful year under the sponsorship of the Society. Few of us realize the tremendous amount of work, effort and talent devoted to the initiation and operation of this project, particularly by Dr. Louis Baldwin and Mrs. Katherine Baldwin. During the past year, the blood bank has been the object of some criticism from outside organizations and individuals, but a recent investigation by an impartial group resulted in a very favorable report and recommendations that the bank be continued under the sponsorship of the Society. The operation of the bank, in spite of some ill-considered criticism, has reflected credit upon the Society and the management of the bank. Certain changes and expansions in the service are contemplated and the united support of the members of our Society will contribute immensely to its continued successful operation.

There is one other agency which is not directly sponsored by the Society, but which draws its advisory board largely from our members to which I would like to call your attention. The Visiting Nurses' Service, under the very able direction of Miss Rogers, was inaugurated a little over a year ago and has rendered an excellent service to the community and the profession in Phoenix. If you have not already utilized this service, may I suggest that you familiarize yourself with what it has to offer? It is worthy of our support, and I urge you to utilize its services to the advantage of both your patient and yourself.

During the past year, the Society has considered and discussed at great length, at times somewhat heatedly and acrimoniously, but finally, we trust, with a commendable degree of harmony and unanimity, the medical service plan sponsored by the Arizona State Medical Association. This is now in the final stages of organization; and at latest reports, its inauguration is scheduled for the near future. Successful operation of this plan can not be accomplished without the sincere cooperation of all members of our profession, and I feel justified in urging your whole-hearted support as the greatest aid in overcoming the inevitable obstacles and possible mistakes which may attend the inauguration of such a venture.

Now, as I relinquish the office of President, it is with mingled feelings of regret, satisfaction and relief—regret that I may not have measured up to my responsibilities and always faithfully carried out the will of the Society and fulfilled the qualifications for President which is stated in the By-Laws, "to be the real head of the profession in the County during the year and to leave the Society in better condition, both as regards scientific attainment and harmony than when he took office"—satisfaction that some definite objectives have been attained and agreement reached upon important controversial steps without seriously disrupting the harmony which prevailed during the administration of my predecessor, Dr. John W. Pennington, who guided the Socoiety so succesesfully for two years during the war—relief in that the responsibilities incumbent upon this high office now rest upon other shoulders and that the society has chosen so wisely one who will represent us creditably and under whose guidance we can go on to ever higher achievement. I am deeply appreciative of the honor which you conferred upon me and my fellow officers; and for them and myself, I bespeak for our worthy successors the same whole hearted support and willing cooperation which you have accorded us.

I wish here to express my very special appreciation to my fellow officers and to the various chairmen and committee members who always responded so willingly and carried out assigned tasks so efficiently. Dr. Thoeny, it is with pleasure I welcome you to the chair and relinquish to you the office of President of the Maricopa County Medical Society.

DISTINGUISHED GUESTS ATTEND THE GILA COUNTY MEDICAL SOCIETY MEETING OF DEC. 17, 1946

The Gila County Medical Society, presided over by Dr. Marcus G. Kelly of Miami, Arizona held its annual meeting at the Cobre Valle Country Club, Tuesday evening, December 17, honoring their wives and distinguished visitors

of the Women's Auxiliary of the Medical Association. Present were: Mrs. Jesse D. Hamer, national president of the Women's Medical Auxiliary of the United States from Phoenix; Mrs. Carl Harris, state treasurer; and Mrs. Thomas H. Bate, vice-president of the State Auxiliary; and Mrs. Harvey S. Faris of Tucson, president of the State Auxiliary. Mrs. Bate and Mrs. Faris were accompanied by their husbands, Dr. Thomas H. Bate and Dr. Harvey S. Faris, who were also guests of the Society.

Also present were Mr. Gerald Craig, superintendent of Gila County Hospital and his wife; and Mr. Guy Ligon, manager of Miami-Inspiration Hospital and his wife.

Members of the Society who attended and their wives were as follows: Dr. and Mrs. Clarence Gunter; Dr. and Mrs. T. C. Harper; Dr. and Mrsr. A. J. Bosse; Dr. and Mrs. Walter M. O'Brien; all of Globe; Dr. and Mrs. John Aarni of Ray, Arizona; Dr. and Mrs. Nelson D. Brayton; Dr. and Mrs. Cyril M. Cron; Dr. and Mrs. Marcus G. Kelly; Dr. and Mrs. Ira E. Harris; Dr. and Mrs. Robert M. Wade; Dr. and Mrs. M. E. Burgess; Dr. and Mrs. Russell R. Nice, all from Miami.

At the annual election of officers which followed the banquet, Dr. M. E. Burgess of Miami was elected President; Dr. A. J. Bosse of Globe, vice-president; Dr. Nelson D. Brayton of Miami, secretary-treasurer.

In the Scientific Session, Dr. Bate of Phoenix, gave a very informative address on the usage of Penicillin in Europe while in the U. S. Army.

Staff Meetings

MARICOPA COUNTY MEDICAL SOCIETY

October 7

1. Precancerous Lesions of the Skin — Dr. Louis G. Jekel.
2. Variations in Treatment of Cancer in Relation to Anatomical Location—Dr. John Foster.
3. A Plea for Earlier Diagnosis in Cancer—Dr. Norman D. Hall.

November 4

1. Recent Advances in General Surgery—Dr. Thomas H. Bate.
2. Recent Advances in Orthopedic Surgery—Dr. Geo. A. Williamson.

January 6

1. Recent Advances in General Medicine—Dr. Kent Thayer.
2. Recent Advances in Cardiovascular Diseases—Dr. Wm. D. King.
3. Recent Advances in Gastrointestinal Disease—Dr. Joseph Bank.

PIMA COUNTY MEDICAL SOCIETY

October 5

1. The Allergic Army—Dr. William Steen.
2. Aberant Pancreas—Dr. Arthur J. Present.
3. Motion Picture Film—"Sulfathalidine in Treatment of Chronic Ulcerative Colitis." (courtesy Sharp and Dohme)

November 8

1. Treatment of Diarrhea—Dr. B. P. Storts.

ST. JOSEPH'S HOSPITAL (Phoenix)

September 25

1. Eosinophilic Hyperplasia or Granuloma—Dr. M. W. Merrill.
2. Clinical Diagnosis and Discussion, opened by—Dr. Geo. A. Williamson and Dr. E. Henry Running.
3. Histopathological Diagnosis and Discussion—Dr. T. T. Frost.
3. X-ray Diagnosis and Therapy—Dr. John Foster.
5. Hypertrophic Gastritis Resembling Neoplasm. A report of two cases with Lantern Slide Demonstration—Dr. Joseph Bank.

October 11

1. An Unusual Ureteral Condition—Dr. John W. Pennington.

Pathological Discussion by Dr. T. T. Frost.

2. Eosinophilic Leukemia—Dr. T. Del Giorno.

Discussion by—Dr. R. H. Stevens.

December 6

1. Case of Diabetes Insipidus with Unusual Etiological Factors—Dr. H. Denninger.

Discussion of a case with special reference to diagnosis and treatment of Diabetes Insipidus—Dr. M. R. Richter.

Pathological Findings—Dr. O. O. Williams.

January 9

1. Case of Thrombocytopenic Purpura with Splenectomy—Dr. Frank J. Milloy.

Discussion of the Surgical Aspects — Dr. Thomas W. Woodman.

Pathological Discussion—Dr. T. T. Frost.

2. Case Presentation and Follow-up on a Case of Epidural Abscess—Dr. M. W. Merrill.

ST. MONICA'S HOSPITAL (Phoenix)
October 16

1. Psychogenic Factors in Two Asthma Cases —Dr. Albert Eckstein.

November 16

1. Non-Specific Urethritis—Dr. Paul Singer.

ST. MARY'S HOSPITAL (Tucson)
July 16

1. Pneumoenteritis, a New Disease?—Dr. F. C. Gregg.

August 20

1. Case Report Thrombocytopenic Purpura—Dr. H. C. Thompson.

September 17

1. Enterogenous Cyst, Case Report—Dr. H. G. Cogswell.

October 15

1. Esophageal Varicies associated with Hemophilic Arthritis—Dr. H. E. Kosanke.

2. Scalenus Anticus Syndrome, bilateral — Dr. Meade Clyne.

GOOD SAMARITAN HOSPITAL (Phoenix)
October 28

1. Anticoagulant Therapy—
Case Presentation—Dr. Haislip
Case Presentation—Dr. Cohen
Clinical Use of Dicumarol and Heparin—Dr. Cohen.
General Discussion.

2. Vomiting in the Newborn, with Case Report—Dr. E. Henry Running.

35—Arizona Medicine

3. Use of Penicillin in Beeswax in Certain Pediatric Conditions—Dr. Carl Holmes.

November 25

1. Pneumonectomy for Bronchogenic Carcinoma with Presentation of Patient—Drs. Kent Thayer and D. W. Melick.

2. Use of Penicillin in Beeswax in Certain Pediatric Conditions—Dr. Carl A. Holmes.

3. Amebiasis, Case Report—Dr. D. B. Haislip.

Clinical Pathological Conferences

Phoenix Clinical Club

A fifty-six-year-old Greek woman was admitted to the hospital complaining of chills and fever and a mass in the left upper quadrant.

Three years before admission the patient entered another hospital because of a painful mass in the left upper quadrant and easy bruising of six months' duration. She had tired easily and had lost about 20 pounds in weight during the preceding year. She also complained of episodes of red urine and a bloody vaginal discharge. Physical examination at that time revealed a small wizened woman who showed evidence of considerable weight loss. There were petechiae on the palate, and a tourniquet test was positive. Several quite large purpuric areas were seen on the extermities, especially on the legs. The liver was felt by one observer two to three finger-breadths below the right costal margin. The spleen extended to 10 cm. below the left costal margin. Hinton and Wassermann tests were negative. The urine was normal. The red-cell count was 3,500,000, with 10.5 gm of hemoglobin and a hematocrit of 30 per cent; the white-cell count was 2100, with 55 per cent neutrophils, of which 10 per cent were band forms, and 42 per cent lymphocytes. The red cells showed mild anisocytosis, and the platelets were markedly reduced, numbering 59,000 per cubic millimeter. A gastric analysis showed no free acid in the fasting specimen and 17 units of total acid after histamine. The icteric index was normal. X-ray examination of the long bones was negative. During the twenty-three hospital days her temperature was 98.6°F. every third day. She was given four x-ray treatments of 65 r each over the splenic area, with no change in the size of the spleen. Five days after x-ray treatment she was discharged improved.

The patient had taken quinine irregularly once or twice a week for about a year, with some relief. During the five months before admission to this hospital she experienced regular attacks of chills and fever, occurring every seventy-two hours between 6:00 and 7:00 p.m. A backache in the upper lumbar region was present at all times of the day and had persisted since the onset of the present illness. For approximately two months before admission her appetite had

been poor, and she had some exertional dyspnea and orthopnea and a slight cough.

The patient was born in Greece and had lived there for eighteen years. She came to the United States thirty-eight years previously and had worked in a cotton mill in Massachusettts. She was married at the age of twenty-six and had had two full-term babies, both delivered by cesarean secton. One infant was stillborn, and the other died at fourteen days. Until the present illness she had been in good health. She remembered no infections or other diseases.

Physical examination revealed an emaciated, chronically ill-appearing woman in no distress but for occasional stabs of pain in the left upper quadrant and epigastrium, which were induced by moving about. The skin was warm and dry and had a slight yellow tinge. The peripheral lymph nodes were not remarkable. There were many vericose veins over the legs and thighs, and fairly prominent superficial veins over the lower chest and abdomen. There were no ecchymoses. There was slight pitting edema of the legs. The left pupil measured 2 mm. in diameter and was fixed and slightly irregular. The neck veins pulsated in the erect and supine positions. The lung fields were clear and hyperrosonant. The left heart border was at the midclavicular line. A Grade 2 blowing systolic murmur was heard over the entire precordium and was loudest at the left sternal border in the fourth interspace. The spleen was firm, smooth and mobile; it extended into the left lower quadrant and a small distance across the midline at the level of the umbilicus. The liver edge was sharp and extended three fingerbreadths below the costal margin. There were no ascitos. Vaginal and rectal examinations were negative. The tendon reflexes of the lower extremities were absent.

The temperature was 99.6°F., the pulse 90, and the respirations 20. The blood pressure was 140 systolic, 70 diastolic.

On admission the blood contained 3,200,000 red cells, with 7 gm. of hemoglobin and a hematocrit of 26 per cent. The white-cell count was 2400, with 61 per cent neutrophils and 35 per cent lymphocytes. A reticulocyte count was 1.2 per cent. The platelets were slightly reduced in number. The bleeding time, clotting time and clot retraction were normal. The urine was straw-colored, with a specific gravity of 1.018, and gave a test for albumin; the sediment contained an occasional red cell and hyaline cast. A catheterized specimen show no growth on culture. A stool was dark, formed and guaiac negative. The sedimentation rate and a van den Bergh test were normal. The serum nonprotein nitrogen was 36 mg. per 100 cc., and the protein 5.58 gm., the albumin-globulin ratio being 1.71. The phosphorus was 4.5 mg. per 100 cc., and the alkaline phosphatase 2.9 Bodansky units. A Hinton test and a spinal fluid Wassermann test were negative. A cephalin flocculation test was in forty-eight hours, and the prothrombin time was 21 seconds (normal, 18 to 20 seconds). A bromsulfalein test showed 30 per cent retention forty-five minutes after the injection of 5 mg. per kilogram. An electrocardiogram was normal. A phenolsulfonopthalein test showed 55 per cent excretion in two hours, and the urine concentrated readily to a specific gravity of 1,020. A lumbar puncture yielded normal findings.

X-ray examinations of the chest and abdomen showed the left diaphragm to be elevated, with the heart in a transverse position. The aorta was tortuous, with some calcification in the arch. A large mass extended down from the left upper quadrant to overlie the sacrum and the left iliac wing, displacing the colon downward and the stomach to the right. The left kidney outline was seen through the mass. A gastro-intestinal series was negative. No esophageal varices were seen.

At 5:30 p.m. on the second hospital day the patient had a severe chill. At 7:00 p.m., the temperature rapidly rose to a peak of 106°F., and during the following six hours gradually returned to normal. The pulse and respiratory curves closely followed the temperature curve. Four similar episodes of chills and fever occurred during the following two weeks at regular seventy-two-hour intervals. Blood cultures taken during the chills and during the peak of the fever showed no growth, and blood smears taken at the same time showed no parasites. Because the spleen was firm and presumably fibretic, a splenic puncture was deemed safe and was performed.

Discussant: Dr. Ben P. Frissell.

Given a patient whose presenting complaints are chills and fever and a mass in the left upper quadrant and having resided for a good many years in a malaria infested district, my

"14 Days to 14 Years"

When should rickets prophylaxis begin, and how long should it be continued? Let's look at the cold figures and the recommendations of leading investigators . . . . . (*over*) . . . . . .

"14 Days to 14 Years"

The Cold Figures

. . . clinical rickets in 37% of 9-months-and-less infants.[1]

. . . signs of rickets in 90% of a group of pre-school children.[2]

. . . histologic evidence of rickets in 46.5% of a group aged 2 to 14, with a peak of 62% in the 10 to 11 year group.[3]

. . . dental cavities twice as frequent in 5 to 15-year old children not on vitamin D as in those receiving it.[4]

The Recommendations

. . . "by the tenth or twelfth day of life, vitamin D in a concentrated form . . . should be started."[5]

. . . administer vitamin D prophylaxis "... from the second week . . . until maturity . . . "[6]

. . . "the addition of vitamin D to the diet is just as important for older children . . . "[7]

The standard by which the biologic activity of all antirachitic agents is evaluated is cod liver oil. White's Cod Liver Oil Concentrate provides the Natural vitamins A and D of time-proved cod liver oil itself, in three palatable, stable, convenient dosage forms well suited for adequate protective administration from 14 days to at least 14 years.

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6. Kugelmass, E. N.: Newer Nutrition in Pediatric Practice, pp. 651-653, Lippincott, Philadelphia, 1940.

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first impression was that our patient was suffering from an infection by plasmodium malariea. However, I can assure you that since my first impression I have had considerable misgivings about this diagnosis and likewise more than a little difficulty in substituting another equally as good.

In the second paragraph of the protocol we are given ample evidence that we are dealing with some type of extensive involvement of the reticulo-endothelial system. Our patient, three years before our present consideration, had an enlarged spleen and liver, a rather extreme anemia of the microcytic type, a marked leukopenia with a preponderance of lymphocytes in the differential count, and a marked reduction in the platelet level with purpura and mucous membrane bleeding as outstanding symptoms. These symptoms were of six months duration at the time of the initial hospital observation, dating the apparent onset of the patient's symptoms to about 3½ years before he present consideration. It is notable that at that time she had a very unusual and characterisic temperature curve—being found to be afebrile except for rhythmic rises regularly on the 3d day. Clinical improvement resulted rather promptly, according to the protocol, from relatively small doses of x-ray over the spleen without making any change in the size of the spleen. It would be assumed that the improvement was in purpura or, perhaps, pain in the region of the spleen.

Considering these initial symptoms, several possible diagnoses can be eliminated. 1—That the patient had pupura of some time is self-evident, but we are able to rule out so-called purpura hemorrhagica or idiopathic thrombocytopenic purpura from the fact that the leukocyte count is markedly surpressed, for in this condition leukocytosis is found. 2—Likewise, aplastic anemia of the primary type is unlikely for with the hematocrit reading of 30% we are obviously dealing with a microcytic, rather than a macrocytic anemia. 3—The various leukemias, which certainly should occupy a very prominent place in differential diagnosis of a case of this type, are, from the first, placed decidedly in the background by the character of the differential count. Even in the so-called aleukemic phase, which would be the only acceptable phase of this disease which would be at all tenable with a WBC of 2,100, the differential count should still show the characteristic pathological cells typical of leukemia.

Our patient had taken quinine irregularly for about a year before her present hospital admission. Five months before this admission, and we assume for the first time, she began having regular attacks of chills and fever occurring regularly, in fact very regularly, at 72-hour intervals. She continued to have lumbar backache throughout a period of three years from the onset of her illness. General nutrition is poor but apparently shows no marked change from the initial examination. At this time there are no hemorrhagic tendencies. The splenic enlargement is somewhat increased, being described as extending into the left lower quadrant and across the mid line at the level of the umbilicus. The liver is still palpable 3 fgb. below the costal margin—the edge described as sharp and the contour otherwise not mentioned. There is no ascites, but moderate edema of the lower extremities is now in evidence. Varicose veins of both lower extremities are described. The veins over the lower trunk are described as prominent. These, of course, could be due to weight loss, or may indicate obstruction of the portal system.

The blood picture is essentially unchanged with the exception that there is no recorded platelet count. However, with normal bleeding and clot retraction time, it would be assumed that the platelet count probably was within normal limits. Urine shows albumin, few hyaline casts. The NPN is within normal limits. Blood proteins, which were not mentioned originally, are slightly below the normal level. The albumin-globulin ratio, likewise, shows a slight but definite shift. Blood and spinal fluid serology are reported as negative. Two liver function tests are definitely positive. Prothrombin time is reported a shade above the upper limit of normal. Regular pattern of fever and chills is observed over a period of two weeks before a diagnosis is presumably made by splenic puncture. Repeated blood cultures and blood smears during this time failed to reveal any evidence of parasites or bacterial growth.

Diseases affecting or involving the so-called reticulo-endothelial system are numerous, and and by no means will all of them even be mentioned in this discussion. The structures of this system have to do primarily with blood formation and phagocytosis, and tissues of this class form components of the spleen, liver, lymph-

nodes, bone marrow and linings of the blood vessels. Pathological conditions effecting these structures have been variously classified but, to say theleast, none too clearly or logically. For the purposes of this discussion we will cover only a few of the more likely diagnostic possibilities and shall merely segregate these conditions into non-infectious and infectious diseases.

The general group of leukemias has already been discussed and I feel definitely eliminated. Various so-called lymphomas—including lymphosarcoma, lymphblastoma and Hodgkin's disease, certainly deserve consideration in the differential diagnosis of this case.

Lymphosarcoma, generally conceded as a malignancy of lymphatic tissues, is progressively fatal but responds very readily to x-ray treatment. It is more likely to involve the lymphnodes proper, in various areas, than the spleen, although it may involve this organ as well as other tissues, including the bone marrow and liver. The WBC in this condition is usually elevated with an increase in PMN cells; anemia is of the secondary type and occurs rather late. Fever is usually low grade and of irregular type. There is a definite tendency to spread from one group of lymphatic structures to another. In the case under consideration, there are no evidences of involvement of any peripheral lymphnodes.

Lymphoblastoma, or the so-called follicular type of malignant lymphoma, quite similar in many respects to lymphosarcoma, likewise considered malignant in nature, is more readily susceptible to x-ray treatment but usually fatal —in 50% of cases terminating in less than 5 years. There is an absence of fever generally and likewise a minimum amount of cachexia. There is usually no anemia except as a result of radiation. Here again, lymphnode groups are primarily involved.

Hodgkin's disease, occurs at any age, usually in the 30-40 year range. Although the progression of the disease is very rapid with fatal termination, chronic forms may go on for a good many years. The spleen is usually enlarged in this condition (60-70%) and the liver is likewise enlarged (50%). Retroperitoneal and mediastinal lymphnodes are most commonly involved. The blood picture shows a progressive anemia but usually no change in the leukocyte count—there is occasionally an eosinophilia and likewise an increase in monocytes. The characteristic Pel-Ebstein fever of this disease differs very definitely from the fever pattern exhibited in the case under consideration. This pattern is characterized by gradual build-up of fever over a period of three or four days to a level of 103-104% with a plateau at this level for another three or four days, then a gradual drop to normal, with cycles of this type running every ten to fourteen days. Severe pruritis is a common symptom in this disease and is not evidenced in the case under consideration.

Gaucher's disease, a rare condition usually occurring in younger individuals and primarily limited to the Jewish race, generally considered as a disturbance in fat metabolism involving the reticulo-endothelial system, is characterized by a chronic anemia and marked splenomegaly. The onset is usually before the 13th year and runs a chronic course up to about 20 years. Anemia is progressive—hemorrhagic tendencies are quite common. The skin usually has a brownish discoloration and there has been frequently described a peculiar wedge-shaped, yellowish pigmentation of the conjunctivae. There are definite changes in long bones which are evidenced on x-ray examination. Fever is not usual in this disease. This fact in addition to the usual date of onset, the racial characteristics and absence of bone changes, is against this diagnosis.

Non-lipoid histiocytosis is a rare condition involving the reticulo-endothelial system. It is variously classed as being very closely related to aleukemic phase of monocytic leukemia, or perhaps neoplastic in origin. The malignant course is characteristic. The involvement of lymphnodes is more prominent than the spleen —anemia, leukopenia and thrombocytopenia are usually present. Fever is of an irregular septic type. Jaundice is quite common. Diagnosis can be made only by biopsy; the differential blood count is not diagnostic.

Banti's disease (if there be such a thing) the so-called splenic anemia described by Banti in 1894, is characterized by gross enlargement of the spleen, progressive anemia and leukopenia and an eventual development of portal cirrhosis of the liver with ascites. Splenic enlargement occurs early and may last for years before any other symptoms develop. Anemia is of the secondary type. Hemorrhage from the stomach is

common. This disease usually occurs in young individuals and is generally classed as a disease of childhood. The liver in the stage of cirrhosis is decreased in size. Fever is not generally a part of this syndrome. The frequent gastro-intestinal hemorrhage is not noted in our case.

Cirrhosis of the liver of the portal type must be considered in this case. This condition may be extremely low grade, may occur without jaundice or without any notable increase in the icteric index. Anemia is usually present and of the macrocytic type but may be, on some occasions, microcytic — particularly when complicated by other conditions such as hemorrhage. Low blood proteins with reversal of the AG ratio are characteristic. Positive cephalin flocculation test, which is an indication of alteration in the serum proteins, is likewise presumptive evidence of portal cirrhosis. The fact that malaria is frequently mentioned as a causative factor in the etiology of portal cirrhosis makes this condition a definite possibility in completing the diagnostic picture of the case at hand.

Tumors (malignant) of both liver and spleen should be mentioned. In the case of the liver, the principle primary tumor is carcinoma. The spleen is not usually enlarged. There is usually a leukocytosis. Fever is of irregular, low grade pattern Jaundice occurs in at least 50% of the cases. Clinically the symptoms point to the liver, whereas in our case we have no symptoms referable to the liver area. As to tumors of the spleen, sarcoma is most common. Except for the splenic enlargement and pain there is nothing to indicate such a diagnosis.

Inflammatory or infectious conditions. In this group tuberculosis must, of course, be considered. Primary tuberculosis of the spleen does occur, but rarely. Involvement of the spleen and liver in a process of miliary tuberculosis is rather common. There is no clinical or x-ray evidence of tuberculosis in this case.

Syphilis, another old stand-by, if it were to explain the symptomatology in this case would most likely be of congenital type. The age of the patient is, of course, against this diagnosis, as is the finding on repeated occasions of negative blood and spinal fluid serology.

There remain three inflammatory conditions which I consider worthy of mention. I favor the primary diagnosis of an infectious process over a neoplastic or metabolic disease. The basis for this assumption admittedly hangs by a thin thread but is established on the definite regular pattern of intermittent fever which is found throughout this case from beginning to end. The three conditions which I consider from this point are (1) malaria, (2) kala azar and (3) histoplasmosis. Nowhere in the literature have I been able to find mention of a disease which duplicates the particular fever-chill pattern described in this case which is so typical of quartan malaria. This disease continues to be one of humanity's chief scourges. There are many fertile areas which still remain practically uninhabitable because of its presence. It is estimated that at least 2,000,000 people die annually from this infection, and in the United States 1,000,000 cases are reported and about 5,000 deaths occur annually. True enough, there are several loopholes in this diagnosis. 1—Our Greek woman, living in Massachusetts at least 18 years after arriving in this country from Greece, supposedly having no recollection of malaria or other notable infection prior to that time, would be considered fairly well outside the so-called malaria belt. However, it is not beyond the realm of possibility, or probability, that this patient may have returned to her native land of Greece and there contracted her malarial infection. 2—Likewise, the initial symptoms are not usual for this type of malaria. 3—It is certainly not usual to have a case go for three and a half years before a diagnosis is established and then by splenic puncture. However, it is a well-known fact that Quinine, in insufficient and irregular doses, may not only obscure the diagnostic criterion in this disease and render it refractory to treatment, but may also be a definite factor in suppression of bone marrow activity. Assuming this case had been treated thusly before our original studies, we would be in a much better position to accept the initial symptoms of purpura, marked anemia, leukopenia, etc. Certainly from the point of the initial hospital admission there is nothing about the clinical protocol which could not be completely explained on the basis of this diagnosis—for malaria which is either untreated or proved refractory to treatment produces pathological changes in all of the tissues with which we are concerned in this instance.

Splenomegaly, for instance, is one of the most frequent physical findings in malaria and ex-

treme enlargement of the spleen may very well occur. The liver is likewise enlarged—this enlargement is usually accompanied by hepatitis. In chronic, uncontrolled cases these changes may result in cirrhosis, and certainly the so-called "bilious" symptoms of malaria are legend. The finding of evidence of liver involvement in this case can therefore, I feel, be explained on this basis.

The kidneys likewise are involved in a good percentage of cases of malaria—the process being described usually as a subacute nephritis. We find in our case a 3+ albumin, a few red cells and scattered casts.

The blood picture in malaria is typically that of anemic and leukopenia. There are multiple factors in the production of anemia, which may be either macrocytic or microcytic in character. The parasites in malaria invade and destroy blood cells. There is inhibition of the bone marrow due to attack on the young reticulocytes in these areas. Activities of the phagocytic cells of the reticulo-endothelial system is increased and the spleen through its enlargement has tremendously increased phagocytic power.

Kala-azar is a disease which for a long time was confused with malaria in certain parts of the world where both conditions re endemic. This condition is very seldom seen in the United States except in rare cases of patients having contracted the disease on foreign soil and returning to their native country. Several instances of this type have been reported in literature since the recent war. From the standpoint of symptomatology and pathology this disease produces a much more complete clinical pattern than does malaria, with few exceptions. Kala-azar is endemic, among other areas, in the regions bordering on the Mediterranean Sea—which, of course, includes Greece. Our patient could have, therefore, contracted this condition after returning to her native haunts for a visit. The disease typically is found, however, in much younger individuals—particularly in that part of the globe it occurs almost entirely in children. The onset of the disease varies a great deal from an acute febrile onset to a slow, insidious process with the initial symptoms practically duplicating those described in the initial paragraphs of the protocol. There is widespread involvement of the bone marrow by the so-called Leishman bodies, which are characteristic of this disease, producing a very severe degree of anemia leukopenia and thrombocytopenia. Although the diagnosis can be established by blood smears on some occasions, the usual way to make the diagnosis is by splenic puncture. Blood protein levels are not markedly diminished but the albumin-globulin ratio is disturbed. This fact is frequently utilized in a diagnostic test for this disease. The fever curve in kala-azar is described as being variable—ranging from irregular fever to intermittent type with chills. However, reviewing as many cases as I could locate in the recent literature of this disease, as well as covering the standard literature on the subject, I was unable to find a single case duplicating the reported type of chill and fever. For that reason, I favor malaria over kala-azar as a diagnostic possibility, however, it is with great reluctance that I do so.

Histoplasmosis, first described by Darling in 1906, is another inflammatory disease involving the reticulo-endothelial system and deserves consideration in this case. This disease, unlike kala-azar to which it bears much similarity, is found in the United States not infrequently. In fact, until 1945, 79 cases had been described in the United States, a much larger number than reported in the rest of the world during the same time interval. Symptoms of the disease are protean—there is usually an irregular type of fever, loss of weight, anemia, leukopenia, general debility, marked splenomegaly and hepatomegaly and usually lymphadenapathy. Some cases show respiratory and skin lesions, while others are chiefly visceral in distribution. Diagnosis may be made in life from the blood smears and blood cultures, using dextrose agar medium, but most cases so far reported have been diagnosed at autopsy. There has been no treatment discovered for the disease and the outcome is uniformly fatal. This disease has frequently been confused with Hodgkin's and is more common in the male than the female, occurs at any age—usually after 10 years, and is usually fatal within one year. Anemia is of the microcytic type and blood counts show a preponderance of lymphocytic cells. The causative organism is an yeast-like fungus which invades the cells of the reticulo-endothelial system. There is rather marked tendency to ulcer formation of the mucous membranes and superficial lymphnodes are enlarged in over 50% of the cases.

In conclusion, it is my choice to stand on the diagnosis of malaria with strong convictions that it far from completely explains the clinical picture in question and, fool-heartedly enough, on the fact that it particularly solves the problem of the pattern of regular chills and fever.

CLINICAL DIAGNOSIS

Quartan malaria

DR. DAMESHEK'S DIAGNOSIS

Quartan malaria

ANATOMICAL DIAGNOSIS

Quartan malaria

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN The intern and resident staff and the visiting men all looked at the smears, thick and thin, and were unable to find any malarial parasites. The material that I received from the spleen was composed almost entirely of histiocytes. I looked carefully for parasites and was unable to find any. There were no hemosiderin granules, which one usually finds in a malarial spleen. Dr. Volweiler took the slides to the Department of Tropical Medicine at the Harvard Medical School, and Dr. Geiman kindly examined them. We shall hear from him now.

DR. CASTLEMAN: The intern and resident films of the splenic puncture to me for examination and told me the history of the case. The history immediately interested me greatly because of the number of cases of so-called "transfusion malaria" that have occurred at various hospitls in Boston. Blood films from the patients hospitals in Boston. Blood films from the patients are brought to us for examination from time to time. Although we find positive films in these patients, the donors of the blood seldom gibe a history of recent clinical malaria and no malarial parasites are found.

Since this patient was born in Greece and was having periodic chills and fever every seventy-two hours, quartan malaria seemed to be the first possibility, at least in my experience; the patient could have obtained the infection a number of years previously. The films from the splenic puncture were examined for ten minutes and parasites were not found at that time. I might say that the films were examined for ten minutes, which is in excess of the usual five minutes for the study of such films.

I asked Dr. Volweiler to make films, both thick and thin, at intervals during a period of several days with the hope that we might catch some of the parasites during their asexual cycle in the peripheral blood. These films were brought to me, and the thick film taken at 4:00 p.m. on February 6 yielded one parasite, which was an unmistakable half-grown schizont, with typical cytoplasm, chromatin and pigment. With that one parasite, I went back through the splenic film, examining it from one end to the other, and fortunately another parasite, a segmenter, was found that determined the exact species, Plasmodium malariae. The examination of additional blood films, made at intervals, produced a total of ten unmistakable and two doubtful parasites. The result of prolonged microscopical study gives you an idea of the scarcity of malaria parasites in this patient. It is outstanding that such a low concentration of parasies could be responsible for clinical malaria.

I thought you might be interested in a case reported by Shute which emphasizes the same point. The case seen by Shute and this case demonstrate that the parasite of quartan malaria can persist in a patient for long periods of time and eventually give rise to a relapse of malaria. At the Chiidren's Hospital, Boston, one of the donors of blood for a case of transfusion had been in this country thirty-seven years without ever having clinical malaria. What the basis for this latency is we do not know, but new information is being accumulated which suggests that there are tissue stages of the parasite in the spleen or other reticuloendothelial tissue that are the reservoir of organisms for his small dribble of parasites into the circulating blood and the origin of parasites that cause relapsing malaria.

DR. CASTLEMAN: I might add that the patient hs received Atabrin and is now free from chills and fever.

CHICAGO CONFERENCES

Rural Health, Medical Service, Medical Education and Licensure

The ANNUAL NATIONAL CONFERENCES ON RURAL HEALTH, MEDICAL SERVICE and MEDICAL EDUCATION AND LICENSURE were held in the Palmer House, Chicago, February 7 to 11th inclusive. For the sake of brevity and because of considerable overlapping in the discussions, a summary of all three of these meetings will be embodied in this report.

Report on the Second Annual National Conference on Medical Service, and the Forty-third Annual Congress on Medical Education and Licensure.

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THE CONFERENCE ON RURAL HEALTH concerned itself with many problems, not the least of which is the present day lack of adequate medical care for our rural population and the seriousness of this problem in the future. That this is a major problem in many rural areas was clearly demonstrated. More and more, as the trend towards specialization in the various fields of medicine progresses, there is a diminution in the number of doctors willing to engage in rural practice where work is hard, practice is general, and there is frequently a lack of hospital facilities and the modern clinical and laboratory aids which are seemingly essential to the present day graduate and the modern practice of medicine.

That the lack of medical attention is having and will continue to have a definite effect on the health of a large percentage of our population was demonstrated in Army induction examinations during World War II, when compiled statistics and results showed that the inductee from rural areas suffered from preventable and uncorrected disabilities to a far greater extent than the men from urban regions. It was repeatedly pointed out that many smaller communities lack any medical attention, and that the average age of the man in rural general practice grows higher year by year. Many of our rural practitioners today are advanced in years and as they retire or die, frequently they are not replaced.

During the entire Conference there was no disagreement as to the need for more adequate rural medical care. When it came to a discussion, however, as to how best to meet this problem, undercurrents of dissension began to be noted. The principal conflict was between leaders of various farm groups and the medical profession, if conflict it could be called. Apparently there is a growing sentiment among leaders of farm groups that some sort of subsidization of medical care will have to be undertaken and in the reports of some representatives, the desire for national compulsory health insurance was very thinly veiled. Others among the farm groups are pushing the development of medical cooperatives with the control retained by the lay organization. Others are in favor of group practice and the establishment of rural health centers and the providing of more adequate rural hospital facilities. Active opposition

flared up in the Conference to the legislation already passed in a number of States which vests 51% or over of the control of voluntary prepayment medical insurance plans in the hands of the doctors and medical organization, the term "restrictive" being applied to these laws.

Other suggested remedies for meeting the critical rural health problem concerned the development of State Health Councils to survey and outline plans to improve rural medical care. Many States have active Councils composed of representatives of the medical and dental professions, public health officials, lay organizations, and other groups with an active interest in social problems. The formation of these Councils in all States was strongly urged.

VOLUNTARY PREPALMENT MEDICAL INSURANCE PLANS came in for a great deal of discussion and it was the general consensus of opinion that they are helping and will continue to aid more in the future as they expand their activities and as more plans are put into operation.

From several sources came a plea for a broader interne training program to fit men for general practice and to encourage them to practice in rural areas. Some medical schools have already incorporated this type of internship training for men interested in becoming general practitioners, Colorado specifically having been trying this for the past several years with encouraging results.

There was general agreement that rural hospital facilities must be greatly improved and that clinical and laboratory facilities must be made readily available if present day graduates are to be attracted to and held in rural medical practice.

Despite the undercurrent of dissension apparent at times, the conference closed with the majority of those attending feeling that many problems had been brought out into the open and that with increased interest and cooperation between the various farm groups and the medical profession as a whole, these problems will be gradually satisfactorily solved and continued improvement made in the critical rural health situation.

THE TWENTIETH ANNUAL CONFERENCE ON MEDICAL SERVICE was concern-

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CHICAGO CONFERENCES

(Continued from Page 155)

ed with a variety of subjects. Dr. Joseph S. Lawrence of the Council on Medical Service of the American Medical Association, gave an able preview of what the 80th Congress is attempting to do and pointed out that there is very little likelihood that much legislation pertaining to naional health will be forthcoming between now and July, with taxation, the budget, and labor legislation holding priority for action at the present time.

Dr. Herman E. Hilleboe of the United States Public Health Service gave an excellent discussion of the recently enacted Hospital Construction Act, pointing out the various phases of the bill, how it will operate, and what may be expected from it during the next five years. He urged early action on the part of each State to initiate a survey of hospital needs within the State and prepare a master plan of hospital construction for submission to the United States Public Health Service, which has been designated by Congress as the agency to carry out the provisions of the Act. The Federal Hospital Council, an appointive body, is working in close cooperation with the Public Health Service as an advisory body on all aspects of the plan. It was pointed out that Federal funds are available to help each State survey its needs and prepare its master plan. It was also pointed out that State legislatures must pass enabling Acts and Licensure Laws to qualify for aid under the provisions of the law. This has been done in some States and is pending in others. Dr. Hilleboe pointed out that hospitals constructed under the Act are to be in no way under Federal control and that the States must assume responsibility after the funds for survey and construction have been allotted. There is no stated limit as to bed capacity of the hospitals to be built, the size depending on the need. It was urged that careful study and planning be given to this program to prevent the construction of what in the future might prove to be unnecessary and inadequately manned institutions.

Dr. Paul B. Magnusson of Chicago, who has been aiding the Veterans Administration in its medical program, made a strong plea for cooperation between Veteran Administration doctors and the general medical profession. He also urged a program of medical schooling and interne training that will increase the number of good general practitioners.

In the open forum discussions the problem of the great trend toward specialization was discussed at length. It was apparent that medical educators, leading men in the profession, and the men heading the specialty Boards are alarmed at the present tendency for every graduate to want to specialize and pass one or another of the various Board examinations. This tendency toward specialization was given a great boost by the war, as it was the policy of the Military to give higher rank to men with specialty Boards and many younger medical officers resolved to obtain specialty training. The fears were expressed that this continued tendency may undermine the cornerstone of American medicine, promote extensive group practice, further concentrate doctors in urban areas, raise the cost of medical care to the public and in time, because of this latter factor, speed the enactment of national compulsory health legislation.

The general conclusions drawn were that more men must be given training fitting them for general practice, that more accent must be placed on the worth of the general man, and that rural communities be provided with facilities that will encourage good men to settle and practice in these areas. The practice of some hospitals in various sections of the country to admit only men with Specialty Boards to their staffs was strongly decried and it was urged that general men be granted hospital privileges commensurate with their abilities.

The two day CONGRESS ON MEDICAL EDUCATION AND LICENSURE proved to be in many ways a continuation of discussions on problems that were brought out in the two previous conferences. The residency training program in Veterans Hospitals was outlined and discussed. These do not now affect Arizona, only Veterans Hospitals close to medical schools being used at the present time.

Dr. Edward L. Bortz, Chairman of the American Medical Association Committee on National Emergency Medical Service, gave a forceful review of medical problems in World War II. He pointed out that because of inadequate reparation and the emergenny character of medical mobilization, much misuse of medical manpower occurred with an over abundance of doctors in

(Continued on Page 159)

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CHICAGO CONFERENCES

(Continued from Page 157)

the military service and a serious lack for civilian needs. He outlined the probable needs when and if another war comes and the plans under way to prevent a repetition of previous errors.

Dr. Thomas Parran, Surgeon-General, United States Public Health Service, gave a talk on the Hospital Construction Act closely paralelling the one delivered by Dr. Herman E. Hilleboe.

The final day of the session was devoted to discussions pertaining to legislation pending or recently enacted in various States and the problems of licensure. The question of adoption of a uniform basic science law was brought up and discussed by Dr. Orin E. Madison, President of the American Association of Basic Science Boards. Dr. Madison outlined a model basic science law and urged its nation-wide adoption. The law he outlined closely resembles Arizona's Basic Science Act in all its major components.

The medical migration since the war to warmer, more equitable climates, particularly California, was ably discussed by Dr. Frederick M. Scatena of Sacramento, California, Secretary of their Board of Medical Examiners. He outlined California's procedure which includes a personal oral examination of each applicant. In 1946 forty-eight per cent of the applicants passed the California Board.

There was considerable open forum discussion among members of the various State Licensing Boards, with a free interchange of ideas as to the best way to evaluate applicants.

The following recommendations are made with reference to the problems discussed at these conferences:

First—That the State Association concern itself with the formation of a State Health Council and cooperate in the formation of local Councils and participate actively in those already formed.

Second—That the State Association take an active interest in the plans for participation in the National Hospital Construction Act.

Third—That the State Association consider a broader, more extensive public relations program to embrace not only relations between medical men and the lay public, but to strive actively to improve the relations within the ranks of the profession itself. This came in for considerable unofficial discussion at these conferences and in view of our pending participation in a voluntary prepayment medical insurance program this problem assumes even greater prominence.

Respectfully submitted,
M. W. MERRILL, M. D.

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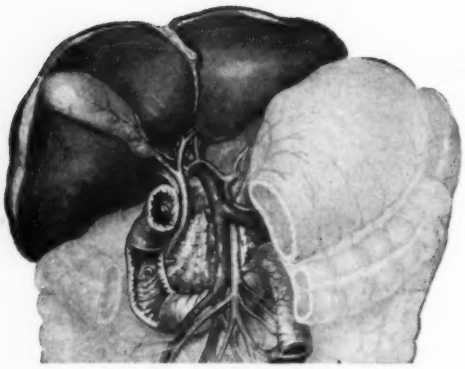

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MEDICO-LEGAL SECTION

BEFORE THE INDUSTRIAL COMMISSION OF ARIZONA

In the matter of the application of Dr. W. A. Seydler for recognition by the Industrial Commission of Arizona of a Hospital.

FINDINGS OF FACT AND CONCLUSION OF LAW AND ORDER

Dr. W. A. Seydler, having filed application with The Industrial Commission of Arizona for recognition of an osteopathically owned or controlled hospital located at First Street and McDowell Road, Phoenix, Arizona, for the hospitalization, care, and treatment of workmen's compensation cases at said hospital under the care of osteopathic physicians and surgeons; and,

The Commission having considered the matter, now takes judicial notice of certain matters herein set forth, and makes Findings of Fact and Conclusions of Law, as follows:

Judicial Notice

1. The Commission takes judicial notice of the fact that approximatetly 95,000 injuries to workmen insured under the Workmen's Compensation Law occurred in Arizona in the year 1945; that of these cases, more than 20,000 cases involved loss of time resulting from said accidents.

2. That the primary purpose of the Industrial Commission Act is to minimize the extent of injuries through prompt and adequate medical care, and to rehabilitate the injured workman and to compensate the workman or his dependents, as far as money may do so, within the framework of the Compensation Law.

3. That the Commission, through the vast number of cases it has handled, knows that even the best trained and most careful medical men frequently know little concerning the basic problems which cause disability for work, and that even the best of medical care available frequently leaves injured workmen permanently, or to a large extent, incapacitated for work.

4. That injuries frequently produce psychic reactions in injured workmen which result in disability where no disability ordinarily should occur.

5. That the Commission is primarily concerned with injured workmen and their rehabilita-

(Continueh on Page 162)

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BEFORE THE INDUSTRIAL COMMISSION

(Continued from Page 160)

tion; secondly, with seeing that the cost of rehabilitating and caring for injured workmen is kept at the lowest cost to the employers who must pay the insurance premiums; and third, that as to all others who render service, that they be paid the fair value for such services, but recognize the principle that no profession has any vested interest in the care or treatment of injured men, or securing compensation benefits for them.

Findings of Fact

1. That all employers subject to the Compensation Law who insure under the provisions of Section 56-932, to provide compensation benefits, are required, in addition thereto, under the provisions of Section 56-939, to furnish first aid, and under the provisions of Section 56-940, to provide medical and hospital care and accident benefits; or to insure the payment of such benefits by insuring the same with the private insurance carrier or in the accident benefit fund, in which latter event, the insurance carrier or the accident benefit fund stands in his stead for the payment of said benefits, and may assert any rights or powers the employer may have; Karns v. Ind. Com., 50 Ariz. 466, 73 Pac. (2d) 104.

2. That under the provisions of Section 56-966, the duty is imposed upon every physician selected by an employer, or an insurance carrier, to treat an injured workman; or every physician called by an injured workman, to immediately report said accident to the Industrial Commission.

3. That the Industrial Commission is vested with the power, and the duty, under Section 56-964 and Section 56-966, to regulate the fees of said physicians to see that said injured workman secures proper and adequate medical care; and if it deems it advisable or necessary, either upon its own motion, or upon the motion of an insurance carrier, or the employee, to remove said workman from the care of his attending physician and place him under care that will be adequate for his needs.

4. That the Commission has adopted rules pursuant to law in this regard; and has followed a policy of insuring spelialized care to injured workmen; that it has frequently had occasion to remove patients from the care of in-

(Continued on Page 164)

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Fourth, and most important, all persons concerned must have a thorough knowledge of the handling and operation of oxygen cylinders, regulators, and oxygen administering apparatus.

All the above are as near to you as your telephone, **call 3-2149.**

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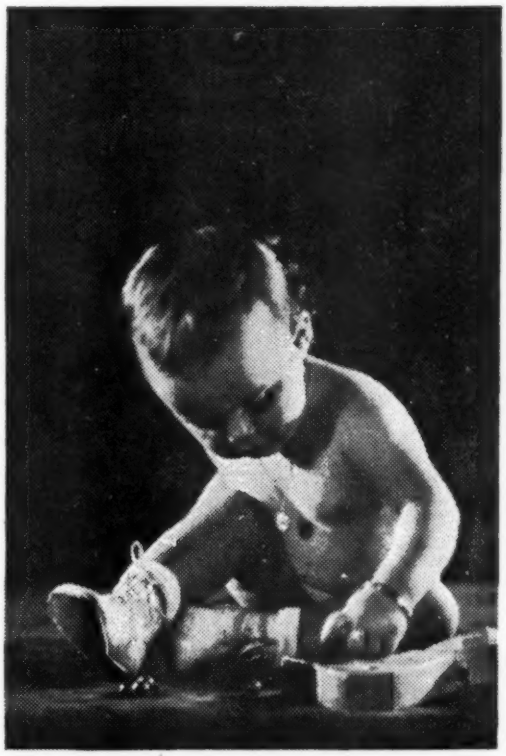
"To maintain Borden leadership in scientific and sanitary requirements, to deliver this vital food to you when you need it, regardless of difficulties. Finally, to bring Borden's to you at a price that will enable millions to enjoy milk that can be depended upon ... always."



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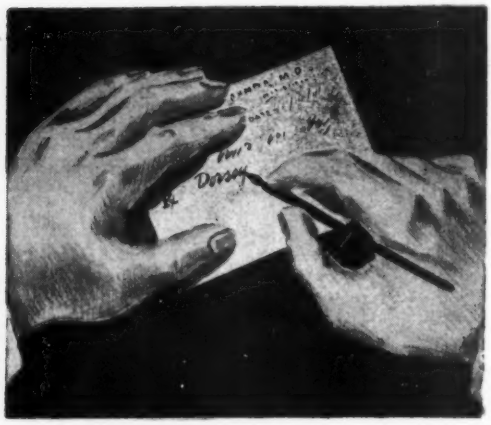

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Chances are most physicians have never visited the pharmaceutical laboratories where the medications they use routinely are manufactured. You yourself, perhaps could not name the scientific staff or describe the methods followed in your favorite drug house.

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BEFORE THE INDUSTRIAL COMMISSION

(Continued from Page 162)

ternists, or general surgeons, and place such patients under the care of orthopedists or urologists; and frequently has occasion to take patients from the care of surgeons and orthopedists and place them under the care of physiatrists.

5. That by reason of such power, and duty, and its experience with many thousands of cases, the Commission must, of necessity, be governed by the needs of each individual case, and has, and now continues, to refuse to recognize the blanket right of any doctor hospital, physician, or practitioner of the medical arts to any vested right in the treatment of any injured man.

6. That the Commission, through its experience in the past, knows the inadequacies of many of the branches of the healing arts licensed to practice in the State of Arizona; that the Commission has no preconceived prejudices either for or against any of the healing arts, but that it does know that, for instance, manupulation of a Pott's fracture by a practitioner in one of the healing arts, on the theory of reducing a sprain materially aggravated the injury of such workman.

7. That the Commission knows that the reading and diagnosis of injuries through x-ray is a highly technical and skilled branch of the medical profession; that the Commission, at all times, insists that x-rays be taken and read under the supervision of a radiologist who is a member of the College of Radiology, and not by a general practitioner, even though he may be licensed and qualified by reason of his license to read x-rays; in other words, the Commission must, of necessity, in its duty to the injured workman, determine the degree of skill required to read and diagnose injuries from x-rays in justice to the injured workman and in justice to the employer who must pay the premium for the treatment of the injured workman, and any errors in judgment in reading x-rays; and the Commission takes judicial notice that there is a wide variance of opinion among even the most skilled radiologists concerning the diagnosis of injuries from x-ray pictures.

8. That the Commission presently has no adequate knowledge of the capacity, or sufficiency, of the osteopathic hospital or the doctors who

(Continued on Page 166)

tests prove

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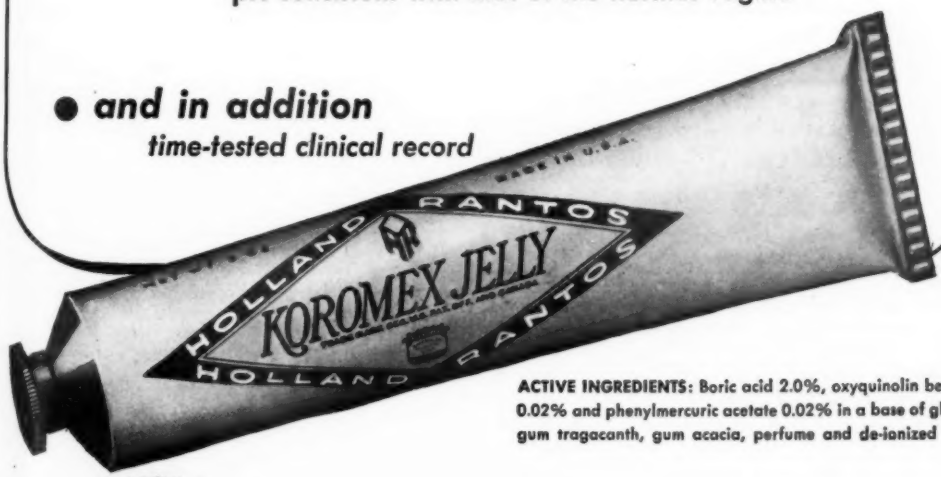

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BEFORE THE INDUSTRIAL COMMISSION

(Continued from Page 164)

constitute the staff thereof, to treat workman's compensation cases.

9. The Commission takes judicial knowledge of Section 67-2115, A.C.A. 1939, but finds that it is not required as a matter of law, to accept the qualifications of said doctors or hospital for the care and treatment of workmen's compensation cases, and finds that under the provisions of the Workmen's Compensation Law and the Arizona Occupational Disease Disability Law, the determination of the fitness, training and capacity of persons selected to diagnose ca for, and treat workmen's compensation cases, vested in the Industrial Commission of Arizon subject, however, to the requirement that i actions in the premises be an exercise of judici discretion.

NOW, THEREFORE, IT IS ORDERED th the request of Dr. W. A. Seydler that the ost opathic physicians and the osteopathic hospit located at First Street and McDowell Rd., Pho nix, Arizona, for blanket approval for treatme of workmen's Compensation cases, is denied.

IT IS FURTHER ORDERED that said oste opathic hospital be approved for the receipt an treatment of first aid cases only; that cases re ceived at said hospital are to be reported to th Industrial Commission with request for further authorization to furnish accident benefits to in jured workmen. The Commission reserves the exclusive right to determine whether further treatment of each idividual case is authorized at said hospital, or by any of the osteopathic physicians or surgeons attending such case or cases.

THE INDUSTRIAL COMMISSIOON
OF ARIZONA.
By Ray Gilbert
Earl G. Rooks
Fred E. Edwards
Commissioners.

Dated at Phoenix, Arizona,
this 5th day of December, 1946.

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Book Reviews

"The Story of Human Birth:" by Dr. Alan Frank Guttmacher. Published by Penguin Books, Inc., and Reprinted by arrangement with The Viking Press. Price: 25 cents.

The layman, for whom this book was written, will obtain from this book reliable information of the folklore, the history, and the scientific facts of pregnancy and birth.

Dr. Guttmacher begins with a vivid word picture of a sixteenth-century delivery by a barber-surgeon. Then he portrays every phase of the development of the foetus in utero, of the prenatal care of the mother, of spontaneous birth, of normal convalescence from childbirth and complications after childbirth. Lastly, attention is turned "to the permanent result, the baby." Written for the "dazed" father, an attempt is made to answer the questions he would like to ask the doctor about the new born baby. L. J.

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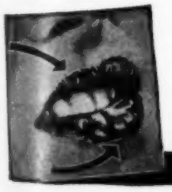

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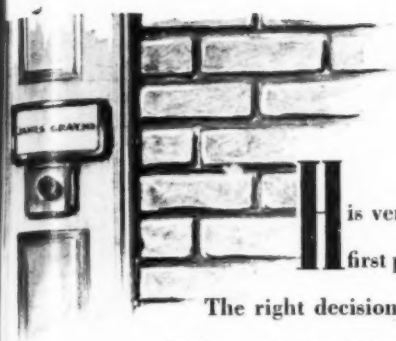

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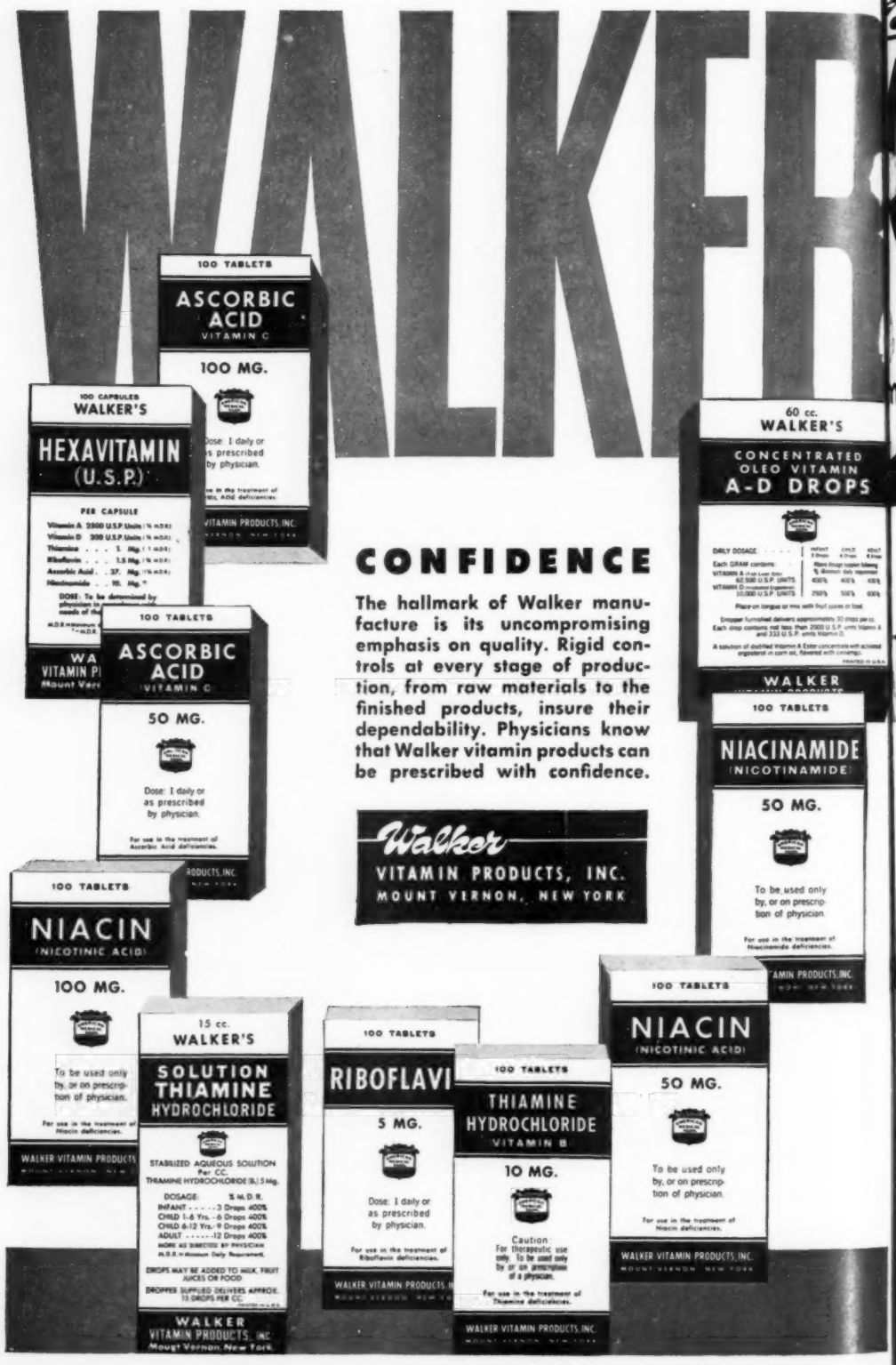
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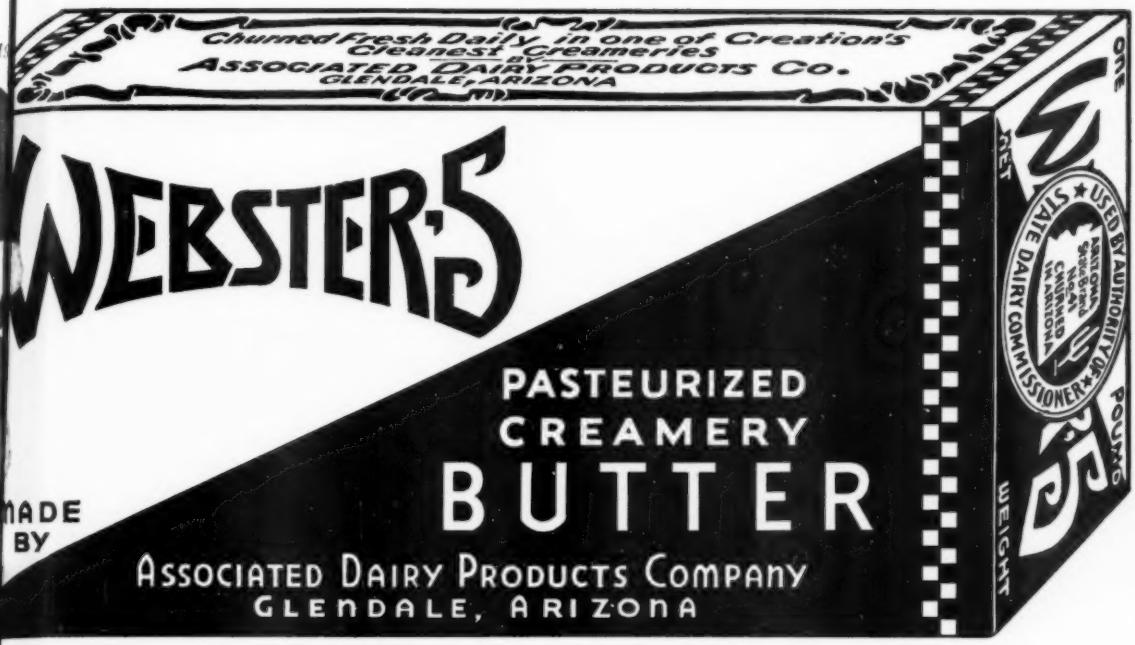

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